

NEUROCOGNITIVE PROCESSES IN DISORDERED EATING

By

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Abstract

The overall aim of this thesis was to better understand the specific cognitive and neural mechanisms that may serve as risk factors to the development of disordered eating behaviour. In Chapter 2, findings are suggestive of a novel mechanism guiding attention to food cues in overweight/obesity through working memory. Differential attentional processing of food cues was also found to be a predictor of weight gain at one-year follow-up. In Chapter 3, a systematic review of the evidence for an association between Attention Deficit Hyperactivity Disorder (ADHD) and disordered eating, suggests a moderate strength of between ADHD and disordered eating; a framework for future research was proposed to guide future studies on ADHD and disordered eating. Chapter 4 aimed to address some of the research gaps outlined in Chapter 3. Notably, in two independent studies, findings provide the first evidence for a direct relationship between inattentive symptoms of ADHD and binge/disinhibited eating behaviour. In Chapter 5, an experimental design was employed to investigate eating behaviour in ADHD using laboratory measures, in conjunction with self-report measures, along with performance-based tasks to assess specific cognitive constructs, and neural correlates of eating behaviour. This model can also be used to the study of other mental disorders associated with disordered eating behaviour. Overall, this thesis provides novel and theoretical insights into the role of attention in guiding eating behaviour. The findings can inform future research and may have implications for the management and treatment of individuals with overweight/obesity, ADHD and/or disordered eating.

“The real meaning of enlightenment is to gaze with undimmed eyes on all darkness.”

(Nikos Kazantzakis)

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Ethical Approval

The British Psychological Society (BPS) guidelines for human research ethics were adhered to in the design and conduct of the research studies that form this thesis. Ethical approval was granted by the Science, Technology, Engineering and Mathematics Ethical Review Committee at the University of Birmingham. The Study described in Chapter 1 has received additional approval from the National Research Ethics Service (NRES), NRES Committee West Midlands – The Black Country.

Statement of Authorship

This research was conducted as part of a PhD studentship funded in the UK by the Biotechnology and Biological Sciences Research Council (BBSRC) in collaboration with P1vital. Chapters 2-5 contain material that has been published, submitted to or prepared for publication in peer-reviewed journals. Consequently, each chapter has been written as a stand-alone paper. Repetition of material has been avoided where possible; however, there may be some overlap between chapters. The authorship of each chapter (published, submitted or unpublished) indicates collaborative work. All authors were involved in the design of the study. I collected and analysed the data in all of the chapters under the supervision of Suzanne Higgs, Pia Rotshtein and Colin Dourish. All chapters were written by me with edits/comments provided by Suzanne Higgs, Pia Rotshtein and Colin Dourish. The authors thank Darshna Patel, Zoe Crocker, Tor Davey and Isabel Ereira for assistance with the data collection for Study 2, Chapter 4 and Louise Simeonov for assistance with the data collection for the Study presented in Chapter 5.

Chapter 1: General Introduction

1.1 Obesity and Overweight

Overweight and obesity are defined as abnormal or excessive fat accumulation that may impair health. Body Mass Index (BMI) is the most commonly used measure to classify overweight and obesity levels in adults, and is defined as a person's weight in kilograms divided by the square of their height in meters (kg/m^2). For adults, the World Health Organization (WHO) defines overweight as a $\text{BMI} \geq 25 \text{ kg/m}^2$ and obesity as a $\text{BMI} \geq 30 \text{ kg/m}^2$. Although, BMI is a useful tool to assess overweight and obesity at a population level, as it is the same for both sexes and for all ages, it should be considered as an approximate measure as it may not correspond to the same degree of fatness in different individuals (WHO, 2016). Waist circumference or waist-hip ratio (i.e. the waist circumference divided by the hip circumference) have been suggested as better predictors for obesity-associated health risks (e.g. diabetes, obesity-associated dyslipidaemia) than BMI, as they correspond better to accumulation of abdominal fat, which can vary considerably within a narrow range of BMI (Balkau et al., 2006; Czernichow et al., 2011; Esmailzadeh, Mirmiran, & Azizi, 2004).

Overweight and obesity are an increasing major global health concern. According to global health observatory data during the period between 1980 and 2014, the world prevalence of obesity more than doubled. In 2014, more than 1.9 billion adults over 18 years of age were overweight (38% of men and 39% of women), of these over 600 million were obese (11% of men and 15% of women). Forty-two million children under 5 years of age were overweight or obese in 2013 (WHO, 2016). In the UK, there are currently around 15 million people classified as obese and this is projected to rise to 26 million by 2030 (Wang, McPherson, Marsh, Gortmaker, & Brown, 2011).

Obesity is associated with a range of concomitant diseases, such as cardiovascular

diseases (CVD), hypertension, Type 2 diabetes mellitus (T2D) and cancer (Finucane et al., 2011) that reduce quality of life, and the life expectancy of severely obese individuals by an estimated 5–12 years (Finkelstein, Brown, Wragg, Allaire, & Hoerger, 2010). Furthermore, obesity increases the financial burden on health services and in the UK: it has been estimated that the health problems associated with overweight or obesity cost the NHS more than £5 billion annually (Scarborough et al., 2011). Therefore, obesity and its consequences present a clear public health challenge.

Existing weight-loss therapies although often effective in the short-term, have a low success rate in the long-term and weight regain is a major problem (Mancini & Melo, 2017). Therefore, it is of paramount importance to determine risk factors (e.g. genetic, behavioral) that may predispose certain individuals to weight gain so that potential health problems may be reduced or prevented.

1.2 Eating Disorders (EDs) and Disordered Eating

Eating disorders (EDs) are severe chronic mental health disorders that are associated with negative health outcomes and have the highest mortality among psychiatric disorders (BEAT, 2015). The Diagnostic and Statistical Manual of Mental Disorders (5th ed.; DSM–5; American Psychiatric Association [APA], 2013), categorizes three primary EDs: anorexia nervosa (AN), bulimia nervosa (BN), and binge eating disorder (BED). Anorexia Nervosa (AN) is marked by a distorted body image, excessive dieting that leads to severe weight loss, and a pathological fear of becoming fat. Bulimia nervosa (BN) is characterised by frequent episodes of binge eating followed by inappropriate behaviours such as self-induced vomiting, inappropriate use of laxatives or diuretics, or excessive exercise to avoid weight gain. Binge Eating Disorder (BED), which was recognised as a separate category of ED in DSM-5, is characterised by recurring episodes of eating significantly more food in a short period of time

than most people would eat under similar circumstances, with episodes marked by feelings of lack of control. By definition, in contrast to BN, self-induced vomiting and laxative misuse are not present or occur only occasionally in BED. The diagnostic criteria for these eating disorders can be found in Table 1.

Although the recorded ED prevalence rate is around 0.5–3%, depending on the specific ED diagnosis, disordered eating symptomatology in the general population has been found to be as high as 12% (Nagl et al., 2016). Disordered eating refers to patterns of eating behaviour that deviate from normal but do not meet all of the criteria for a clinical diagnosis of an ED. Milder representations of eating pathology, such as fear of fatness, body dissatisfaction, unhealthy weight control practises, obsessive thinking about food and loss of control over eating need to be considered because of the associated psychopathology, but also because of the associated risk for development of full-syndrome eating disorders (Goldschmidt, Aspen, Sinton, Tanofsky-Kraff, & Wilfley, 2008).

Individuals suffering from EDs rarely seek professional help due to fear of gaining weight, which can make treatment very difficult (BEAT, 2015). Therefore, identification of risk factors for disordered eating in general population samples may have a significant public health impact, and may aid in both the prevention and treatment of EDs.

Table 1

DSM-5 Diagnostic Criteria for Anorexia Nervosa, Bulimia Nervosa and Binge Eating Disorder (5th ed.; DSM–5; APA, 2013)

Diagnostic Criteria	
Anorexia Nervosa (AN)	<ul style="list-style-type: none">• Restriction of energy intake leading to a significantly low body weight in the context of age, sex, developmental trajectory, and physical health• Intense fear of gaining weight or becoming fat even though underweight• Disturbance in experience of weight or shape, undue influence of body weight or shape on self-evaluation, or denial of the seriousness of the current low body weight
Bulimia Nervosa (BN)	<ul style="list-style-type: none">• Eating large amounts of food in a discrete time frame• A sense of lack of control over eating during episodes• Recurrent inappropriate compensatory behaviours to prevent weight gain (<i>e.g. purging</i>)• Self-evaluation is unduly influenced by body shape and weight
Binge Eating Disorder (BED)	<ul style="list-style-type: none">• Eating large amounts of food in a discrete time frame• A sense of lack of control over eating during episodes• Associated with three or more of the following: (i) eating more rapidly than normal; (ii) eating until uncomfortably full; (iii) eating large amounts of food while not physically hungry; (iv) eating alone due to embarrassment; (v) feeling disgusted, depressed, or guilty afterward

1.3 An integrated approach to the study of overweight/obesity and EDs/disordered eating

Traditionally, obesity and EDs/disordered eating have been considered as separate conditions, and research has been carried out by different professions, who draw upon different theoretical models and experimental approaches. Obesity often attracts the scientific interest of biological sciences, medicine and nutrition, while EDs are usually studied by mental health professionals, psychiatry and psychology (Hill, 2007). However, epidemiological and longitudinal data provide evidence for increased prevalence rates of EDs/disordered eating among obese individuals, as well as for increased risk of obese individuals to develop EDs/disordered eating behaviour (de Franca, Gigante, & Olinto, 2014; Eddy et al., 2007; Flament et al., 2015; He, Cai, & Fan, 2017; Lebow, Sim, & Kransdorf, 2015; Loth, Wall, Larson, & Neumark-Sztainer, 2015). For example, in a cross-sectional population study of 2097 individuals aged 20-59 years, de Franca et al. (2014) found a high prevalence of binge eating among obese individuals, and He et al. (2017) in a recent meta-analysis found that binge/loss of control over (LOC) eating was prevalent among more than one quarter of children and adolescents with overweight and obesity. Notably, the increasing rates of BED along with the increasing rates of obesity, has led some researchers to suggest that BED may have contributed to the increase in the incidence obesity (Yanovski, 2003). However, obesity has also been reported as a risk factor for EDs/disordered eating. For example, Fairburn, Welch, Doll, Davies, and O'Connor (1997) found that the odds of being obese as a child were three times higher among individuals with bulimia as compared to healthy controls. In addition, evidence from a large population-based survey of adolescents, suggest that overweight adolescents are more likely than their non-overweight peers to engage

in disordered eating behaviours, such as diet pill use, vomiting and laxative use (Boutelle, Neumark-Sztainer, Story, & Resnick, 2002).

Overall, there is evidence that overweight/obesity and EDs/disordered eating, often collectively named as weight-related disorders, are not distinct from each other. Integrated approaches to the study of overweight/obesity and EDs/disordered eating, will enable investigation and identification of common and distinct risk factors for these weight-related disorders, providing opportunities for the development of prevention and treatment interventions that may simultaneously target both conditions.

1.4 Appetite Regulation

A dysregulation of food intake is central to both overweight/obesity and EDs/disordered eating. Therefore, a better understanding of all the factors involved in the control of appetite and food intake is required if new approaches to the treatment of both conditions are to be developed.

1.4.1 Homeostatic control. Over the past 20 years, our understanding of the homeostatic mechanisms involved in the regulation of food intake has significantly improved, through dramatic increase in research of how nutritional information is transmitted to the brain (Berthoud, Munzberg, & Morrison, 2017). A scientific breakthrough that acted as an important driver for further advances was the discovery of the hormone leptin. Leptin is produced predominantly by adipose tissue and conveys information to the brain on the amount of energy stored in fat. Leptin levels in circulation increase proportionally to fat mass, and binding of leptin to its receptors in the brain, results in suppression of appetite and increased energy expenditure (Munzberg & Morrison, 2015).

The hypothalamus is recognised as the main brain region involved in the homeostatic control of food intake, receiving signals from the periphery related to short and long-term

nutritional status, such as leptin, cholecystokinin (CCK), ghrelin, orexin, insulin, and neuropeptide Y (NPY), and through the sensing of nutrients, such as glucose, amino acids and fatty acids (Blouet & Schwartz, 2010; Coll, Farooqi, & O'Rahilly, 2007; Dietrich & Horvath, 2009; van Vliet-Ostaptchouk, Hofker, van der Schouw, Wijmenga, & Onland-Moret, 2009). For example, when food is eaten, hormones including cholecystokinin (CCK) and glucagon-like peptide 1 (GLP-1) are released by the gastrointestinal tract (GI) and this information is transferred to the brain, via the vagus nerve, eventually resulting in cessations of food intake (Dockray, 2012; Turton et al., 1996). Leptin effectively informs the brain as to the status of energy stores throughout the body, providing a signal for long-term nutritional status. An example of a hormone that has metabolic effects opposite to those of leptin is ghrelin, a peptide that is primarily secreted by the stomach. Ghrelin levels peak before each meal to stimulate food intake, and then fall to lower levels immediately upon food consumption (Begg & Woods, 2013; Drazen, Vahl, D'Alessio, Seeley, & Woods, 2006).

The caudal brainstem is another major integrator of information on nutrient ingestion relayed from the gut (Grill & Hayes, 2012). Neurones in the nucleus tractus solitarius (NTS) are responsible for processing multiple nutrient status signals from the periphery and relay output to other regions involved in the control of intake including the hypothalamus (Grill & Hayes, 2012).

1.4.2 Hedonic control. In addition to providing nutritional needs to support normal function, food is also a natural reward. Babies' facial reactions to sweet versus bitter tastes suggest that there is an innate tendency for humans to like sweet tastes and dislike bitter tastes (Scott, 1992). Liking, however is only one component of reward, describing the hedonic response to a food stimulus (Berridge, Robinson, & Aldridge, 2009). Berridge et al. (2009) has proposed that, wanting, and learning are also components of reward. Wanting or incentive

salience, is a type of incentive motivation which is closer to motor action to obtain rewards and learning is used to describe predictive associations and cognitions. Thus, food becomes wanted through a learned process that involves repeated associations between the liked item (e.g. chocolate cake) and the hedonic responses that are evoked due to the consumption of that particular food (Berridge et al., 2009). Although, wanting and liking usually converge, this is not always necessarily the case, which suggests that both components are important in order to understand reward processing (Berridge, 2009b).

Research on the neurobiology of food reward circuitry has linked liking to mu opioid and cannabinoid receptor systems in the brain, and wanting to the mesolimbic dopamine system (Berridge, Ho, Richard, & DiFeliceantonio, 2010). Dopamine has been the most thoroughly investigated neurotransmitter involved in modulating reward. In addition to an involvement in the motivation to perform the behaviors necessary to procure and consume the food or “wanting”, dopamine has further more complex roles in reward (Volkow, Wang, & Baler, 2011). Thus, first exposure to a food reward (or an unexpected reward), increases dopamine release in nucleus accumbens (Norgren, Hajnal, & Mungarndee, 2006). However, with repeated exposure to the food reward, the dopamine response habituates and is gradually transferred to the stimuli associated with the food reward (e.g. the sight of food), which is in turn processed as a predictor of reward (becoming a cue that is conditioned to the reward); the dopamine signal in response to the cue then serves to convey a “reward prediction error” (Diederen et al., 2017; Schultz, 2010, 2016). Cues that become conditioned can have a significant impact on eating behaviour, driving food consumption. For example, imaging studies have shown that exposure to food cues elicits dopamine increases in the striatum that are associated with the desire to eat the food (Volkow et al., 2002).

While homeostatic and hedonic systems are usually discussed as two separate systems,

recent models suggest an important interaction between these two systems to regulate eating behaviour (Berthoud et al., 2017).

1.4.3 Interactions between homeostatic and hedonic control. The idea that homeostatic and hedonic systems interact to regulate eating behaviour is supported by evidence that suggests changes in pleasantness sensations are associated with metabolic state. Thus, in a state of hunger food is highly desired and liked, while under conditions of satiety food is less liked (Berridge et al., 2010). Also, there is evidence to suggest that highly palatable food items can drive overeating even under conditions of satiety (Sclafani & Springer, 1976). Taken together, this suggests metabolic state can influence reward processing, and vice versa in an interactive process (Berthoud et al., 2017). In support of this interaction, the metabolic signals leptin and ghrelin have also been reported to influence reward processing. Leptin administration has been found to decrease activity in the mesolimbic dopamine system of rats and decrease food intake (Fulton et al., 2006; Hommel et al., 2006), while leptin administration in humans with leptin-deficiency reduced reward responses to pictures of food (Farooqi et al., 2007). In contrast, ghrelin administration has been found to increase reward related responses to food stimuli (Malik, McGlone, Bedrossian, & Dagher, 2008).

1.4.4 Cognitive processes involved in appetite regulation. Cognitive processes are also involved in the regulation of eating behaviour. For example, the role of attention and memory in the control of food intake has been shown by numerous studies (for reviews see Higgs, 2008, 2016). Amnesic patients, with hippocampal damage, have been found to eat multiple meals in quick succession, suggesting that memory for recent meals plays a crucial role in determining subsequent food intake (Higgs, Williamson, Rotshtein, & Humphreys, 2008; Rozin, Dow, Moscovitch, & Rajaram, 1998). Experimental research, has also found

that augmentation of memory encoding during attentive eating reduces later snack intake (Higgs & Donohoe, 2011; E. Robinson, Kersbergen, & Higgs, 2014). In contrast, distraction while eating (e.g. watching television or playing a computer game) is associated with increased later snack intake (Higgs & Woodward, 2009; Mittal, Stevenson, Oaten, & Miller, 2011; Oldham-Cooper, Hardman, Nicoll, Rogers, & Brunstrom, 2011), as encoding of the meal is disrupted.

Food cues, due to their motivational value attract attention. However, individual differences have been reported in food cue responsiveness (attentional bias), and attentional bias to food cues has been described as a behavioural factor that may drive overeating behaviour (Berridge, 2009a; Polivy, Herman, & Coelho, 2008). Although, bottom-up (reward-driven) processes have been suggested to be involved in attentional bias (Castellanos et al., 2009; T. E. Robinson & Berridge, 1993), recent evidence supports a role for higher cognitive functions, such as working memory in guiding attention (Higgs, Dolmans, Humphreys, & Rutters, 2015; Higgs, Rutters, Thomas, Naish, & Humphreys, 2012; Rutters, Kumar, Higgs, & Humphreys, 2015). Specifically, holding food related information in working memory, a situation similar to a condition of preoccupation with food thoughts, guides attention to similar stimuli, highlighting the role of working memory in attention allocation.

Although enhanced attentional bias to food cues has been suggested to be linked to overeating behaviour, experimental research linking attentional bias to actual food intake is limited and has reported mixed findings (Nijs, Muris, Euser, & Franken, 2010; Werthmann et al., 2014; Werthmann, Roefs, Nederkoorn, & Jansen, 2013; Werthmann et al., 2011). Long-term goals (e.g. weight-loss) have been suggested to modulate responses to food cues. For example, it has been reported that individuals who take into account longer-term health consequences of eating choices are less likely to respond to a palatable food cue by choosing

to consume that item than individuals who do not take the delayed consequences into account (Hare, Camerer, & Rangel, 2009). These data are suggestive of involvement of higher cognitive control processes to inhibit immediate appetitive response to palatable food cues. Deficits in inhibitory control have been linked to overeating and overweight/obesity (Batterink, Yokum, & Stice, 2010; Braet, Claus, Verbeken, & Van Vlierberghe, 2007; Guerrieri, Nederkoorn, & Jansen, 2007; Nederkoorn, Smulders, Havermans, Roefs, & Jansen, 2006). Furthermore, findings from neuroimaging studies support a role of frontal inhibitory networks in the control of food intake (e.g. dorsolateral prefrontal cortex (DLPFC)). For example, lean individuals, tested in a satiated state (*after consumption of a meal*) compared to a state of hunger, showed increased BOLD activation in the DLPFC, to chocolate and strawberry flavours and pictures, suggesting a significant role of cognitive self-control in satiety and inhibition of further eating once eating has ceased (Thomas et al., 2015). In contrast, obese individuals have been found to show less activation in DLPFC after consumption of a meal, suggesting that diminished inhibitory control may contribute to overeating especially in the absence of hunger (Le et al., 2006; Le et al., 2007).

Taken together, these findings are suggestive of strong influences of higher-level cognitive functions, such as attention, memory and cognitive-control on food intake.

1.5 A Neurocognitive Model of Overweight/Obesity and Disordered Eating

Because of the abundance of food cues in today's environment, it is increasingly important to understand how metabolic signals interact with individual differences in responsiveness to food cues, and the role of higher cognitive processes to guide human eating behaviour. Accumulating evidence supports an important role of higher-level cognitive functions, such as attention, memory and cognitive-control in appetite control, and a model was recently proposed in which cognitive processes interact with metabolic and reward

systems to regulate eating behaviour (Higgs, 2016; Higgs et al., in press). This model may have important implications for treatment of both overweight/obesity and disordered eating, and is central to the present thesis, has informed the design of experimental research, with an overall aim to better understand the specific cognitive and neural mechanisms that may serve as risk factors to the development of disordered eating behaviour.

1.6 Thesis Outline

Chapter 2 investigates reward-related differences in response to food cues between normal-weight and overweight/obese individuals. Specifically, attentional processing of food cues in a state of satiety was studied in a sample of overweight/obese individuals in comparison to normal-weight individuals. The paradigm used to capture attentional processing enabled for first time the investigation of both automatic (bottom-up) capture and top-down guidance of attention to food items via working memory, and testing for differences between overweight/obese and normal-weight subjects. Additional aims of the study were to investigate whether attentional biases to food cues can predict (over)eating behaviour in a laboratory setting, and weight gain (BMI change) at one-year follow-up.

Chapter 3 provides a systematic review of the literature concerning the association between Attention Deficit Hyperactivity Disorder and disordered eating behaviour and presents a framework for future research. Attention Deficit Hyperactivity Disorder (ADHD) is defined as a persistent pattern of inattention and/or hyperactivity/impulsivity that is more frequently displayed and is more severe than is typically observed in individuals at a comparable level of development (American Psychiatric Association, 2013). In the context of the present thesis, ADHD provides a model to study the role of core cognitive processes such as attention and inhibitory control in the regulation of eating. The systematic review aimed to provide an estimate of association between ADHD and disordered eating. Specifically, four

questions were addressed: (1) Is there an association between ADHD symptoms and disordered eating behaviour? (2) Are specific features of ADHD differentially associated with specific types of disordered eating behaviour? (3) Are there factors that affect the direction and/or strength of any relationship between ADHD symptoms and disordered eating (moderators)? (4) Which factors could explain the relationship between ADHD symptoms and disordered eating behaviour (mediators)?

Chapter 4 presents data from two independent studies, which aimed to address some of the research gaps outlined by the systematic review presented in Chapter 3. Specifically, the studies investigated potential associations between the core symptoms of ADHD (inattention and hyperactivity/impulsivity) and disordered eating, including both binge/disinhibited eating and restrictive eating behaviour, and assessed whether negative mood and/or deficits in awareness and reliance on internal hunger/satiety cues mediate these relationships. The potential moderating effects of age, gender, BMI and/or ADHD medication in relationships between the core symptoms of ADHD and disordered eating were also investigated.

Chapter 5 reports on investigation of eating behaviour and associated neural correlates in individuals with symptoms of Attention Deficit Hyperactivity Disorder (ADHD) compared to controls. The study aimed to assess differences in eating behaviour between individuals with high ADHD symptoms and control individuals using laboratory measures of eating behaviour in conjunction with questionnaire measures. Specifically, an experimental procedure was employed that enabled investigation of eating behaviour both in the context of a typical meal (lunch), and also in the absence of hunger (snack), providing a potential behavioural measure of binge eating behaviour. The involvement of specific cognitive constructs (e.g. inhibition) and neural correlates of eating behaviour was also assessed.

Chapter 6 presents a summary of the results of the present research, discusses strengths and limitations, future directions and implications of the findings.

**Chapter 2: Top-Down Guidance of Attention to Food Cues Differs Between Normal-
Weight and Overweight/Obese Individuals and Predicts Weight Gain at One-Year
Follow Up**

2.1 Abstract

The aim of this study was to investigate whether attentional biases towards food cues distinguish overweight/obese from normal-weight participants, and to assess whether attentional biases for food cues can predict subsequent food consumption, as assessed in a laboratory setting, and weight gain over a one-year period. A total of 43 overweight/obese and 49 normal-weight individuals between the ages of 18 and 58 participated in the study. To assess attentional processing of food cues a paradigm that captures both automatic (bottom-up) and top-down guidance of attention via working memory was employed. Eating behaviour was assessed via a bogus taste test. At one-year follow-up participants were invited to come back to the laboratory to assess changes in BMI. Overweight/obese showed greater attentional biases towards food cues than did normal-weight participants, but only in the working memory task, that is when they were holding food related information in working memory. During the priming task, food cues had a greater biasing effect than non-food cues as assessed during the subsequent selection task, but no difference was observed between normal-weight and overweight-obese participants. During the taste test, overweight/obese consumed more of the offered food items than normal-weight participants, but only for the highly palatable food items. Attentional biases towards food cues, when food related information was held in working memory, was found to predict weight gain (BMI change) over a one-year period, after taking into account important possible predictors of BMI change. In conclusion, the current study provides evidence for the first time for a novel mechanism underlying attentional biases in overweight/obese individuals via working memory. The longitudinal association that was observed between the biasing effect of food, when relevant information was held in working memory and weight gain over a one-year period, is suggestive of a causal interplay between these two variables.

2.2 Introduction

Obesity is an increasing major global health concern. In the UK, there are currently around 15 million people classified as obese and this is projected to rise to 26 million by 2030 (Wang, McPherson, Marsh, Gortmaker, & Brown, 2011). Obesity is associated with a range of concomitant diseases (Finucane et al., 2011) that reduce quality of life, and life expectancy of severely obese individuals by an estimated 5–12 years (Finkelstein, Brown, Wragg, Allaire, & Hoerger, 2010). Changes in the food environment of Western societies have been proposed to play a major role in the increased prevalence of obesity, such as the abundant presence of food related stimuli (e.g. food advertisements, food displays) that stimulate food consumption (Werthmann, Jansen, & Roefs, 2015; Westerstorp & Speakman, 2008). However, individual differences have been reported in responsiveness to food cues, and individuals with a high food-cue responsiveness (attentional bias), are assumed to be more vulnerable to overeating and weight gain in today's food-rich environment (Berridge, 2009a; Polivy, Herman, & Coelho, 2008).

Why this is the case is poorly understood, and two main theories have been proposed to explain why motivational objects, such as food cues, can have a strong influence on attention. Based on the incentive-sensitization theory, it has been proposed that food attracts attention due to its rewarding properties in a bottom-up manner, as a consequence of a conditioning process, that is after repeated associations between food cues and a rewarding experience, the cues (e.g. sight of palatable food items) become salient and attract attention (Castellanos et al., 2009; T. E. Robinson & Berridge, 1993), which can drive consumption even in the absence of hunger and subsequently weight gain if the increased energy intake is not compensated for. However, recent evidence suggests that higher level cognitive processes are involved in the guidance of attention to food cues, such that holding food related

information in working memory modulates the extent to which food captures attention; “top–down” modulation of attention (Higgs, Dolmans, Humphreys, & Rutters, 2015; Higgs, Rutters, Thomas, Naish, & Humphreys, 2012; Rutters, Kumar, Higgs, & Humphreys, 2015). Specifically, holding information in working memory causes attention to be involuntarily drawn to similar stimuli in subsequent search displays, even if it is irrelevant to the selection task (Soto, Heinke, Humphreys, & Blanco, 2005; Soto & Humphreys, 2007). Taken together, these findings suggest that attentional biases towards food cues are mediated, at least partly, through working memory as well as through bottom-up processes, providing an alternative mechanism through which attentional bias to food cues could be linked to eating behaviour.

Few studies to date, have investigated attentional processing of food cues in individuals with overweight and obesity, and the findings have been mixed. For example, Braet and Crombez (2003) found greater interference of food words in obese children than normal-weight children, using a modified Stroop test, whereas, Phelan et al. (2011) using the same paradigm, reported no significant differences between obese and healthy weight adults. Moreover, Soetens and Braet (2007), using a different attentional task (e.g. the imbedded word task), found no evidence of attentional bias to food cues in overweight adolescents. Of interest, all of the above-mentioned studies used words, rather than pictorial images of food, as stimuli. Food words might have weaker motivational effects (Simmons, Martin, & Barsalou, 2005; Tiggemann & Kemps, 2005), and thus food pictures may be more likely to reveal attentional biases and should be preferred as stimuli. Castellanos et al. (2009) measuring eye movements during a visual probe task with food and non-food pictures found that obese participants showed greater visual attention to food cues than did normal-weight participants. Similarly, Nijs, Muris, Euser, and Franken (2010) and Werthmann et al. (2011) reported an enhanced initial orientation towards food cues in overweight/obese individuals

compared to normal-weight individuals. However, Loeber et al. (2012) reported no differences in attention allocation towards food-associated stimuli compared with neutral stimuli for obese participants or for normal-weight controls. Indeed, in a critical review of the literature, Field et al. (2016) concluded that the overall evidence that obesity is associated with an enhanced attentional bias toward food cues is weak, and the influence of attentional bias on behaviour may have been overestimated. Although, research has demonstrated that individual differences in attentional bias to food cues were positively related to individual differences in ad libitum food consumption that was measured immediately after assessment of attentional bias (Werthmann et al., 2014; Werthmann, Roefs, Nederkoorn, & Jansen, 2013), findings from studies that specifically assessed differences in attentional processing of food cues between normal-weight and overweight/obese participants, failed to report significant relationships between measures of attentional bias and subsequent food intake (Nijs et al., 2010; Werthmann et al., 2011). In addition, only two studies to date have investigated whether there is an association between attentional bias to food cues and weight gain. Calitri, Pothos, Tapper, Brunstrom, and Rogers (2010), reported that cognitive biases to healthy and unhealthy food words, as assessed via the emotional Stroop, predicted changes in BMI over a one-year period in a student sample, but no effect was found when biases were assessed via the dot probe task. Yokum et al. (2011) in an fMRI study also reported that attentional bias to food images were associated with elevated weight and increased risk for future weight gain in a sample of adolescent girls ranging from lean to obese. Taken together, these findings suggest, that it remains unclear whether overweight/obesity relates to enhanced attentional bias to food cues. In addition, the relationship between attentional bias to food cues and actual eating behaviour is uncertain and further research is required to confirm the predictive value

of attentional bias on weight gain, as well as to investigate the underlying mechanisms through which food can capture attention and influence eating behaviour.

To the best of our knowledge, although, food-related thoughts have been reported to be more predominant among heavier individuals (Israel, Stolmaker, & Andrian, 1985) and a memory bias for food stimuli have been described in overweight and obese individuals (Soetens & Braet, 2007), a specific link between working memory and attentional biases for food cues in overweight/obesity has yet to be investigated. Investigating how working memory may guide attention to food related stimuli in overweight/obesity is important as it will enhance our understanding of how higher level cognitive processes, such as holding food related information in working memory, may affect guidance of attention to food cues. Furthermore, as attentional bias towards food cues may predict future weight gain (Yokum, Ng, & Stice, 2011), a better understanding of the underlying mechanisms may have important implications for treatment and/or prevention of overweight/obesity. Previous research has not investigated the specific effects (*if any*) of bottom-up versus top-down processes in the guidance of attention to food cues in overweight/obesity, and this may partly account for the contradictory findings. This study aimed to investigate attentional biases for food cues in overweight/obese and normal-weight individuals, using naturalistic visual stimuli (that is, images of food) and employing a paradigm that captures both automatic (bottom-up) and top-down guidance of attention.

The paradigm we employed has been described elsewhere in detail (Higgs, Dolmans, Humphreys, & Rutters, 2015; Higgs, Rutters, Thomas, Naish, & Humphreys, 2012), and is based on a procedure used previously to assess attentional guidance (Downing, 2000; Soto, Heinke, Humphreys, & Blanco, 2005). In brief, participants are first presented with a cue, which is either a food or a non-food item, followed by a search display. The cue either has to

be identified (the bottom–up priming condition) or it has to be held in memory for a later memory test (the top–down working memory condition). There are three type of trials: (i) neutral, the initial cue does not reappear in the next search display, or (ii) valid, the initial cue reappears next to the search item or (iii) invalid, the initial cue reappears in the opposite field to the search item. The re-appearing item can capture the participants’ attention, even when it is irrelevant to the subsequent selection task (invalid trials). Interestingly, this re-appearance effect was typically much stronger when the first item was held in working memory than when it was merely identified (in the bottom–up priming condition) and it was stronger for food items than non-food items (Higgs et al., 2015; Higgs et al., 2012; Rutters, Kumar, Higgs, & Humphreys, 2015). In the current study, we used this paradigm to investigate how automatic capture and top–down guidance to food items may differ between overweight/obese and normal-weight individuals. An issue with previous research is that attentional bias has been primarily studied in a state of hunger. However, this could mask increased biases in people with overweight/obesity, and therefore in the current study participants were tested in a satiated state. An additional aim of the study was to investigate whether attentional biases for food cues can predict (over)eating behaviour in a laboratory setting, and weight gain (BMI change) at one-year follow-up. Of note, all of the existing studies that investigated the association between attentional bias and food intake and/or future weight apart from one by Calitri, Pothos, Tapper, Brunstrom, and Rogers (2010) included only female participants, and none assessed both the predictive value of attentional biases for both food intake and weight gain. This is important because the determinants of short- and long- term appetite and body weight regulation might differ (Havel, 2001). In the current study, we assessed both the predictive value of attentional biases for (over)eating and BMI change over a one-year period in a sample of both males and females. The paradigm we employed to assess attentional

biases also allows us to assess for first time the role of top-down guidance of attention in predicting food intake and weight gain.

Based on both theoretical and empirical grounds, we hypothesised that overweight/obese individuals might show both stronger bottom-up priming, if food representations are strongly pre-activated by the sight of food in these individuals, as well as showing greater top-down attentional guidance to food-related stimuli, since overweight/obese individuals may already be preoccupied with food thoughts (Israel et al., 1985; Soetens & Braet, 2007). In contrast, normal-weight individuals might be less responsive to food cues because they are able to suppress food items in working memory. If this were the case then the results could identify a novel mechanism underlying attentional biases in overweight/obese individuals. We also hypothesised that overweight/obese participants would show increased food intake compared to normal-weight participants, and that attentional biases for food cues would predict both food intake, and weight gain over a one-year period.

2.3 Materials and Methods

Participants

Adult men and women, aged 18-60 years old, were recruited through the University of Birmingham and the University Hospitals of Coventry and Warwickshire NHS Trust (UHCW) by posters, emails and mailshots. The sample size for the current study was determined by a power calculation. Based on previous results reported from Werthmann et al. (2011), it was calculated that there will be 80% power to detect a difference of approximately 28% in food intake (with overweight/obese individuals eating more than normal-weight ones), using a one tailed t-test (2 independent means) at the 5% significance level if we recruit 88 participants in total. Therefore, we aimed to recruit 44 normal-weight and 44

overweight/obese individuals. To reduce demand characteristics, the study was advertised as research examining eating habits and memory function. To take part in the study participants were required to be fluent English speakers, as we had no resources for translators or interpreters for this study, and should not report: (i) the presence or a history of a diagnosed eating disorder, a psychiatric, neurological or medical illness, (ii) the presence or a history of tobacco use, drug abuse or the use of any medication, within the past month, that might influence eating behaviour and/or body weight, (iii) the presence of a food intolerance and/or allergy. Participants could choose to take part in exchange for money or course credits. All participants provided informed consent for their participation. The study was approved by the National Research Ethics Service (NRES), NRES Committee West Midlands – The Black Country and the University of Birmingham Research Ethics Committee.

Measures

Eating behaviour. Eating Behaviour was assessed by three separate questionnaires, measuring different aspects of human eating behaviour:

The Three Factor Eating Questionnaire (TFEQ) (Stunkard & Messick, 1985), which is a 51-item self-report questionnaire that measures three dimensions of human eating behaviour: "cognitive restraint of eating" (Factor I - 21 items), "disinhibition" (Factor II - 16 items) and "hunger" (Factor III - 14 items). These subscales have been shown to have good reliability and validity (Stunkard & Messick, 1985). In the present study the Cronbach's alpha was 0.83, 0.76 and 0.75 for the "cognitive restraint of eating", "disinhibition" and "hunger" subscales, respectively.

The Dutch Eating Behaviour Questionnaire (DEBQ) (Van Strien, Frijters, Bergers, & Defares, 1986), which is a 33-item self-report questionnaire with three subscales: "emotional eating" (Factor I - 13 items), "external eating" (Factor I - 10 items) and "dietary

restraint" (Factor I – 10 items). These subscales have been shown to have good reliability and validity. Cronbach's alpha in a sample of 653 women and 517 men ranged from 0.80 to 0.95 across scales and groups (Van Strien et al., 1986). In the present study, the Cronbach's alpha was 0.94, 0.83 and 0.91 for the "emotional eating", "external eating" and "dietary restraint" subscales, respectively.

The Power of Food Scale (PFS) (Lowe et al., 2009), which is a 15-item self-report questionnaire that measures appetitive drive to consume highly palatable food at three levels of food proximity: "food available" (Factor I- 6 items), "food present" (Factor II- 4 items), and "food tasted" (Factor III- 5 items). These subscales have been shown to have good reliability and validity. In a sample of obese subjects (n=1741) and a sample of general population (n= 1275) Cronbach's alpha ranged from 0.81 to 0.91 across scales and samples (Cappelleri et al., 2009). In the present study, the Cronbach's alpha was 0.91, 0.87, 0.88 and 0.75 for the total score, the "food available", the "food present" and the "food tasted" subscales respectively.

Attentional tasks. Participants were asked to complete two versions of the attention task, a priming task (the bottom-up priming condition) and a working memory (WM) task (the top-down WM condition), which were very similar, but differed in the instructions to the participants. In the priming task, participants were asked to identify a cue but not to hold it in memory. In the WM task, participants were asked to hold a cue in memory across the trial in order for it to be matched in a subsequent memory test. Both tasks contained valid, neutral and invalid trials and there was a total of 650 trials for each task. On each trial, participants were presented with either a food or a non-food cue (see Figure 1 & 2). The cue was either a picture of a food item, a household item or a stationery item and 10 different pictures per category were used during both tasks. A trial started with a central fixation cross for 600 ms, followed by a cue for 500 ms. After the cue, a fixation cross appeared for 200–1000 ms

(randomly chosen), followed by the search array, which consisted of a target (a circle) and a distractor (a square) that appeared randomly to the left or right of fixation (see Figure 1 for an example of a trial in the priming and working memory task). Participants had to press 'c' if the circle appeared on the left and 'm' if it appeared on the right. The target and the distractor were each flanked by a picture of a food item, a household or a stationery object. The inter-trial interval was 400 ms. In the working memory task, 20% of the trials ended with a memory probe that followed the search display to check that the participants were performing the task correctly and had remembered the cue as instructed. On the memory probe trials, an item from the same category as the cue appeared for 3000 ms and the participants indicated whether the item was the same or different to the cue. Participants pressed 'c' if the item matched the cue or 'm' if it was different. No memory probes were presented for the priming task; however, in the priming task the cue disappeared after 250 ms on 20% of the trials and a different image appeared in its place. On these trials, participants were required to withhold their response to the search task.

On valid trials, the target was flanked by an image that was the same as the cue and the distractor in the search display was flanked by an image from one of the other cue categories. On invalid trials, the distractor was flanked by an image that was the same as the cue and the target was flanked by an image from one of the other cue categories. On neutral trials, both the target and distractor were flanked by images from categories different from the cue (see Figure 2 for an example of the working memory task, representing food valid, food neutral, and food invalid trials). The trials occurred randomly with equal probability. All pictures were matched on visual characteristics, presented in black and white, sized 480×480 pixels and appeared in the middle of the screen with a black background.

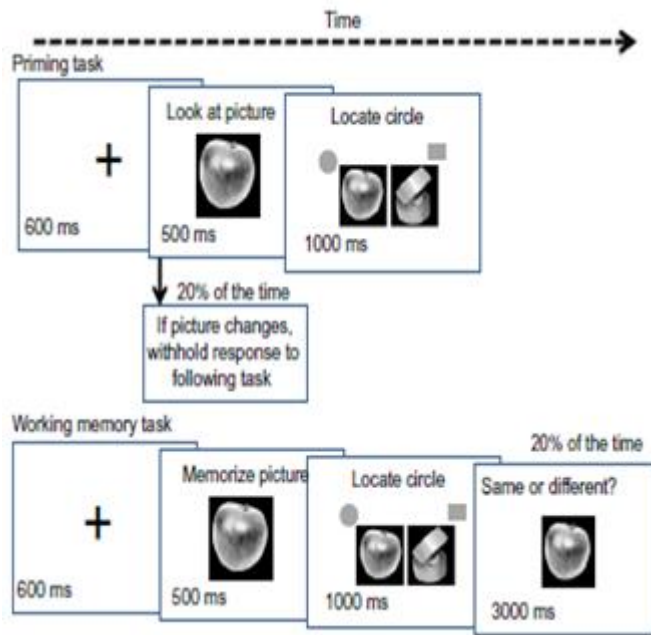


Figure 1. Study design for priming and working memory tasks

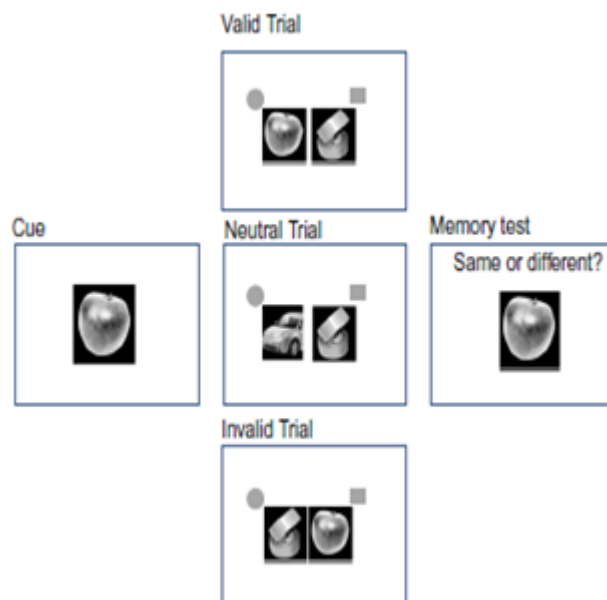


Figure 2. Example of WM task representing a food valid, food neutral and food invalid trial

Taste test. Food consumption was measured by means of a bogus taste test. As previous research is limited by investigating the association of attention to food with the consumption of only highly palatable foods (Nijs et al., 2010; Werthmann et al., 2011), a range of both ‘healthy’ and ‘unhealthy’ food items were offered during the taste test. In this way, it is possible to examine whether attentional bias to food cues is related to a general tendency towards overeating or whether attentional bias to food cues is related only to overconsumption of highly palatable, energy dense foods. During the taste test, participants were instructed to rate four bowls of highly palatable food items (chocolate, chocolate cookies, crisps, salted biscuits) and four bowls of fruit and vegetables (nectarine, melon, cherry tomatoes, cucumber) in terms of their visual attractiveness, smell, and taste. Based on these ratings a liking score was calculated for each food item.

All food items were purchased from Sainsbury’s UK, and participants were exposed to four identical pre-weighed bowls filled with highly palatable food items: ± 255 g of chocolate (529 kcal/100 g), ± 150 g of chocolate cookies (502 kcal/100 g), ± 50 g of crisps (526 kcal/100 g), ± 130 g of salted biscuits (516 kcal/100 g) and four identical pre-weighted bowls filled with fruit and vegetables: ± 250 g of nectarine (45 kcal/100 g), ± 250 g of melon (24 kcal/100 g), ± 250 g of cherry tomatoes (20 kcal/100 g) and ± 225 g of cucumber (10 kcal/100 g). The order of the bowls was fixed, and participants were instructed to taste and rate the food items in a particular order, as consumption order may affect the taste ratings. Each participant was given 30 minutes to complete their ratings and informed that after finishing their ratings they were free to eat as much of the offered items as they liked, since these food items were not going to be used for other participants. A glass of water was also available during the taste test. Consumption was determined by weighing the bowls before and after the "taste test" and the difference in weight from pre- to post- assessment was

converted into calories and used as a measure of food intake. Participants were not aware of the fact that their food intake was weighed afterwards, and that food intake (in kcal) was calculated.

Anxiety and depression.

The Hospital Anxiety and Depression Scale (HADS) (Bjelland, Dahl, Haug, & Neckelmann, 2002), a 14-item self-report questionnaire, which was originally developed by Zigmond and Snaith in 1983 (Zigmond & Snaith, 1983), was used to assess anxiety and depression levels. The HADS utilises a 4-point Likert scale (range 0–3), and includes 7 items for each subscale. The total score is the sum of the 14 items, and for each subscale the score is the sum of the respective seven items (ranging from 0–21). In a review of the literature, Bjelland et al. (2002) found that the HADS is a valid tool to measure anxiety and depression levels in the general population and performs well in screening for the separate dimensions of anxiety and depression. The Cronbach's alpha in the present study was 0.83 and 0.71 for the Anxiety, Depression subscales, respectively.

Procedure

Individuals who expressed an interest in the study and fulfilled the screening criteria were asked to attend the Research Unit on a convenient day to complete the testing session. Testing took place between 09:00 am – 10:15 am, and participants were asked to arrive at the Unit with prior instructions not to consume anything for 9-10 hours (since the previous night). Participants were also advised to avoid exercising the day of testing. Upon arrival at the unit, participants had the chance to ask any questions they may have had about any words or procedures included in the information sheet. If participants were happy to continue, they were asked to sign a consent form. At this point, participants were also asked to report on demographic characteristics (e.g. age, gender) and rate their baseline hunger, fullness, desire to eat and thirst on 100mm Visual Analogue Scales (VAS) anchored by word descriptions at

each end that express two extreme states of the condition (*e.g.* "*Not Hungry at all*", "*Very Hungry*"). Resting Energy Expenditure (REE) and body composition (for the estimation of total body fat) measurements were also obtained at this stage with the use of a metabolic cart and Bod Pod or a TANITA Body Fat Scale, respectively.

Since motivational state is known to affect cognitive biases to food (Mogg, Bradley, Hyare, & Lee, 1998), prior to the beginning of the attentional task, participants were satiated by consuming a meal consisting of a cheese sandwich on white bread and a glass of orange juice (Total Energy Content = 500 kcal). To ensure satiation, participants were asked to take a break of 20 min after the consumption of the meal (Castellanos et al., 2009; Nijs et al., 2010). After the break, appetite ratings were scored again by VAS. Participants were then asked to complete the priming and WM tasks, with an option of a 5 minute break between tasks. The priming and WM tasks were completed in a counterbalanced order. After completion of these tasks, appetite ratings were scored again by VAS. Subsequently, a bogus taste test was performed. Shortly before the taste test, the participants were asked to complete another set of VAS. After the taste test, the participants completed another set of VAS, and finally were left alone in a room to complete the questionnaires on eating behaviour, physical activity, impulsivity and psychological distress. At the end of the experimental session participants were thanked for their time, and reimbursed for participation.

In the 1 year follow up, participants were asked to attend a brief session at the Research Unit, during which body composition measurements were obtained with the use of a Bod Pod or a TANITA Body Fat Scale, as in the initial phase of the study. Participants who were not able or willing to attend the follow-up session were asked whether they were willing to provide a self-report measure of body weight. As body weight measurements may vary depending on the fasting state of the participants', and in order to mimic the experimental

procedures, all participants were instructed to avoid eating and drinking for 9-10 hours (since the previous night) before the assessment of their body weight.

Analysis

Data were analysed using SPSS Statistics 22 (IBM). Independent t-tests were used to compare continuous demographic variables (for example, age) between healthy-weight and overweight/obese groups. The χ^2 -test of independence was used to compare group gender and racial composition using defined categories of White, Black, Asian or Mixed/multiple ethnic groups. To control study-wise Type I error rate, comparisons of eating measure, personality, psychological distress subscales and food liking scores between study groups were initially conducted using multivariate analysis of variance (MANOVA) and were reported using Wilk's Λ statistic. If a multivariate statistically significant difference was found, univariate ANOVAs were used to follow-up MANOVA on the individual subscale scores. Differences in ratings in the hunger, desire to eat, fullness and thirstiness VAS scales were analysed using repeated-measures ANOVAs. One-way ANOVA with group as a factor was used to compare food intake (in kcal) between groups.

Incorrect responses to the search task, memory task, and catch trials, as well as reaction times (RTs) that were ± 3 standard deviations from the mean were removed. Differences in RTs between tasks (WM, priming), trials (valid, neutral, invalid), and cues (food vs. non-food items) were analysed using repeated-measures ANOVAs with group (HW, OW/OB) as a between-subjects factor and Bonferroni corrected follow-up post hoc tests. Additionally, paired t-tests were performed. All tests were two-sided and differences were considered significant at $p < 0.05$. Values are expressed as means and standard deviations (SD).

A regression analysis was conducted in order to examine whether attentional bias predicts food intake and BMI change. Given that, age, gender, physical activity levels, body composition measurements (e.g. body fat, lean weight), eating styles (e.g. dietary restraint, disinhibition), and impulsivity are all possible determinants of food intake and BMI change (Davis, 2009; Finlayson, Cecil, Higgs, Hill, & Hetherington, 2012; Hays & Roberts, 2008; Luke et al., 2006), these factors were included as covariates in assessing the predictive value of attentional biases for food intake and BMI change. Physical activity levels, body composition measurements, eating styles, and impulsivity were all coded as continuous variables.

To assess physical activity levels, the International Physical Activity Questionnaire - Short Version (IPAQ-SF) (Craig et al., 2003), a 7-item self-report questionnaire was used. The IPAQ-SF assesses physical activity during the last 7 days, and the items are structured to provide separate scores on walking, moderate-intensity and vigorous-intensity activity. Computation of the total score for the short form requires summation of the duration (in minutes) and frequency (days) of walking, moderate-intensity and vigorous-intensity activities. Domain specific estimates cannot be estimated. One measure of the volume of activity can be computed by weighting each type of activity by its energy requirements defined in METs to yield a score in MET-minutes. METs are multiples of the resting metabolic rate and a MET-minute is computed by multiplying the MET score of an activity by the minutes performed. An overall total physical activity MET-minutes/week score was calculated. While, IPAQ-SF has been found to overestimate physical activity levels as measured by objective criteria (Lee, Macfarlane, Lam, & Stewart, 2011), it is a more straightforward measure to be used in a laboratory setting.

To assess impulsivity, the Barratt Impulsivity Scale (BIS) (Patton et al., 1995), a 30-item self-report questionnaire, was used. The BIS utilises a 4-point format in which respondents are asked to rate items pertaining to impulsive or non-impulsive (for reverse scored items) behaviours and preferences. Currently, this is the most widely used self-report scale of trait impulsivity; its psychometric properties have been determined in both clinical and non-clinical subjects (Patton, Stanford, & Barratt, 1995; Stanford et al., 2009). In the present study, the total score was used and the Cronbach's alpha was 0.80. Baseline levels of hunger and/or desire to eat, and liking of food are also important predictors of food intake as assessed in a laboratory setting (Robinson et al., 2017), and therefore we also included these variables in the model assessing the predictive value of attentional biases for (over)eating.

2.4 Results

Demographic Characteristics

In total, one hundred and five participants took part in the study between November 2014 and April 2016. Thirteen participants were excluded for the following reasons: (i) following the assessment of height and body weight in our research facilities, 6 participants were excluded, as they were categorised as underweight ($\text{BMI} < 18.5 \text{ Kg/m}^2$), (ii) 5 participants were excluded due to a high error rate on either the catch trials and/or memory trials [$> 3 \text{ SD}$ from the mean], and (iii) 2 participants were excluded as they deviated significantly in their food consumption from the rest of their group values ($> 2\text{SD}$ from their group mean). Therefore, the final sample comprised 92 participants; 49 normal-weight (NW) and 43 overweight/obese (OW/OB) participants. Subject characteristics are summarized in Table 1. As expected, NW and OW/OB participants differed with regard to body composition measurements (see Table 1). The mean BMI was 30.46 kg/m^2 for the OW/OB group and 21.46 kg/m^2 for the NW group. Levene's test indicated unequal variances, $F = 36.16$, $p <$

0.001, so degrees of freedom were adjusted from 90 to 51. Unequal variances were indicated for all the body composition measurements and the adjusted degrees of freedom are shown in Table 1. The OW/OB participants tended (mean = 31.16 years) to be older than the NW participants (mean = 27.27 years), but this difference only approached statistical significance ($p = 0.06$). Groups differed in gender distribution and the NW group included significantly more females than the OW/OB group ($\chi^2(1) = 9.10$, $p < 0.01$). There were no significant differences in race between groups ($p = 0.48$), and the groups did not differ in self-reported physical activity levels during the last 7 days ($p = 0.72$).

Table 1*Baseline Characteristics of Groups*

	Normal-weight (n = 49)		Overweight/Obese (n = 43)			
Demographics	Mean (SD)	Min-Max	Mean (SD)	Min-Max	t (90)	p
Age (years)	27.27 (9.95)	18-53	31.16 (9.82)	18-58	-1.89	.06
	n (%)		n (%)		x²(1)	p
Gender (% female)	43 (87.8)		26 (60.5)		9.10	<.01
Ethnic Background	n (%)		n (%)		x²(3)	p
White/White British	30 (61.2)		23 (53.5)		2.48	.48
Asian/Asian British	15 (30.6)		12 (27.9)			
Black/African/Caribbean /Black British	3 (6.1)		7 (16.3)			
Mixed/multiple ethnic groups	1 (2.0)		1 (2.3)			
Body Composition	Mean (SD)	Min-Max	Mean (SD)	Min-Max	t (51)	p
BMI (Kg/m ²)	21.46 (1.58)	18.7-24.9	30.46 (4.51)	25.0-42.8	-12.43	<.001
					t (71)	
Percent Body Fat (%)	24.25 (6.72)	7.3-37.1	35.2 (10.26)	16.3-51.9	-5.98	<.001
					t (57)	
Lean Weight (kg)	45.10 (6.04)	35.2-62.1	56.62 (13.28)	34.4-90.0	-5.24	<.001
					t (54)	
Resting Metabolic Rate	1395.43 (152.13)	1099- 1781	1775.10 (354.32)	1195-2694	-6.45	<.001
Physical Activity	Mean (SD)	Min-Max	Mean (SD)	Min-Max	t(90)	p
MET-minutes/week	2617.55 (2692.25)	0-13332	2815.76 (2550.63)	0-11688	-0.36	.72

Eating Behaviour, Personality and Psychological Distress

Eating behaviour, personality and psychological distress measures are summarized in Table 2. There was a significant association between group and the TFEQ, $F(3, 88) = 3.78$, $p = 0.02$; Wilk's $\Lambda = 0.89$, partial $\eta^2 = 0.11$, but not on the DEBQ, $F(3, 88) = 1.79$, $p = 0.16$; Wilk's $\Lambda = 0.94$, partial $\eta^2 = 0.06$, the BIS, $F(3, 88) = 0.57$, $p = 0.63$; Wilk's $\Lambda = 0.98$, partial $\eta^2 = 0.02$, PFS, $F(4, 87) = 1.38$, $p = 0.25$; Wilk's $\Lambda = 0.94$, partial $\eta^2 = 0.06$, and the HADS, $F(2, 89) = 1.92$, $p = 0.15$; Wilk's $\Lambda = 0.96$, partial $\eta^2 = 0.04$. Separate, univariate ANOVAs revealed that the multivariate difference for the TFEQ measure resulted from higher scores by the OW/OB subjects than NW subjects on the Disinhibition subscale, but this only approached significance ($p = 0.06$). No statistically significant univariate differences were found for other subscale scores. The only other significant univariate difference related to the Dietary Restraint subscale of the DEBQ; OW/OB subjects scored significantly higher than NW subjects ($p = 0.02$). No statistically significant univariate differences were found for other subscale scores (see Table 2 for details).

Table 2*Summaries of Eating Behaviour, Personality and Psychological Distress Measures by Group*

	Normal-weight		Overweight/Obese		MANOVA		
	(n = 49)		(n = 43)		Between-groups effects		
Personality trait	Mean (SD)	Min-Max	Mean (SD)	Min-Max	F (1,90)	η^2	p
BIS-Total	63.12 (10.50)	47-97	62.25 (8.02)	45-83	0.19	0.00	0.66
BIS-Attention	16.88 (3.98)	10-30	16.09 (2.97)	9-22	1.12	0.01	0.29
BIS-Motor	23.02 (4.44)	16-35	23.09 (3.76)	14-34	0.01	0.00	0.93
BIS-Non-planning	23.22 (4.41)	12-37	23.07 (4.04)	16-31	0.03	0.00	0.86
Eating Behaviour	Mean (SD)	Min-Max	Mean (SD)	Min-Max	F (1,90)	η^2	p
TFEQ-Restraint	8.45 (4.84)	1-19	9.72 (4.33)	1-18	1.74	0.02	0.19
TFEQ-Disinhibition	6.47 (3.31)	1-14	7.81 (3.35)	1-14	3.73	0.04	0.06
TFEQ-Hunger	5.69 (3.14)	1-12	4.88 (2.70)	0-12	1.73	0.02	0.19
DEBQ- Dietary Restraint	2.48 (0.87)	0.9-4.2	2.87 (0.73)	1.5-4.5	5.25	0.06	0.02
DEBQ -Emotional Eating	2.21 (0.95)	0.9-4.9	2.22 (1.02)	0.7-4.3	0.001	0.00	0.98
DEBQ -External Eating	3.12 (0.63)	1.8-4.5	3.08 (0.61)	2.0-4.5	0.07	0.00	0.79
PFS-Total	2.74 (0.81)	1.4-4.3	2.81 (0.78)	1.4-4.4	0.14	0.00	0.71
PFS-Food Available	2.26 (0.94)	1.0-4.2	2.33 (0.96)	1.0-4.5	0.14	0.00	0.71
PFS-Food Present	3.11 (1.08)	1.0-5.0	3.05 (0.96)	1.0-5.0	0.09	0.00	0.76
PFS-Food Tasted	3.03 (0.87)	1.6-5.0	3.18 (0.71)	1.4-4.6	0.78	0.01	0.38
Psychological Distress	Mean (SD)	Min-Max	Mean (SD)	Min-Max	F (1,90)	η^2	p
HADS - Anxiety	7.37 (4.05)	1-16	6.16 (3.68)	0-15	2.20	0.02	0.14
HADS - Depression	3.45 (2.81)	0-12	3.70 (2.86)	0-12	0.18	0.00	0.68

Appetite Ratings

Ratings in the hunger, desire to eat, fullness and thirstiness VAS scales by group are shown in Table 3. Upon arrival at the Unit, and before the consumption of the offered meal, the participants were relatively hungry and thirsty, as expected after an overnight fast. There was an immediate significant decrease in thirstiness ($F(1,90) = 145.60$; $p < 0.001$, $\eta^2 = 0.62$), hunger ($F(1,90) = 181.84$; $p < 0.001$, $\eta^2 = 0.67$) and desire to eat ($F(1,90) = 199.11$; $p < 0.001$, $\eta^2 = 0.69$), and an immediate significant increase in fullness ($F(1,90) = 274.10$; $p < 0.001$, $\eta^2 = 0.75$) after the consumption of the offered meal. No significant time x group interactions were observed, suggesting that NW and OW/OB participants were equally sated before the completion of the computer task. Before the taste test, hunger ($F(1,90) = 56.52$; $p < 0.001$, $\eta^2 = 0.39$), desire to eat ($F(1,90) = 58.06$; $p < 0.001$, $\eta^2 = 0.39$), and thirstiness ($F(1,90) = 77.26$; $p < 0.001$, $\eta^2 = 0.46$) levels increased again significantly compared to the after-meal ratings, and ratings of fullness decreased significantly ($F(1,90) = 44.55$; $p < 0.001$, $\eta^2 = 0.33$). No significant time x group interactions were observed, suggesting that NW and OW/OB participants were equally hungry before the taste test. After the taste test, hunger ($F(1,90) = 101.88$; $p < 0.001$, $\eta^2 = 0.53$), desire to eat ($F(1,90) = 170.74$; $p < 0.001$, $\eta^2 = 0.66$), and thirstiness ($F(1,90) = 142.10$; $p < 0.001$, $\eta^2 = 0.61$) levels decreased as expected compared to the pre-taste test ratings, and ratings of fullness increased significantly ($F(1,90) = 191.82$; $p < 0.001$, $\eta^2 = 0.68$). No significant time x group interactions were observed, suggesting that NW and OW/OB participants were equally sated after the taste test.

Table 3*Mean Subjective Hunger, Fullness, Desire to Eat and Thirstiness Ratings by Group*

		Normal-weight	Overweight/Obese
		(n = 49)	(n = 43)
VAS Ratings		Mean (SD)	Mean (SD)
Before Meal	Hunger	62.18 (24.39)	50.42 (26.56)
	Fullness	19.80 (22.32)	23.44 (18.29)
	Desire to Eat	67.74 (21.31)	56.09 (24.66)
	Thirstiness	77.20 (22.60)	73.07 (25.36)
After Meal	Hunger	23.86 (20.73)	19.54 (19.26)
	Fullness	61.06 (22.90)	64.02 (23.39)
	Desire to Eat	30.47 (22.99)	26.09 (20.48)
	Thirstiness	42.94 (24.65)	41.44 (28.14)
Before Taste Test	Hunger	43.25 (24.26)	31.98 (25.08)
	Fullness	44.08 (22.71)	49.00 (26.50)
	Desire to Eat	49.16 (22.95)	42.61 (27.34)
	Thirstiness	67.27 (25.71)	61.02 (25.22)
After Taste Test	Hunger	11.49 (15.77)	10.21 (17.83)
	Fullness	80.28 (19.76)	82.98 (16.80)
	Desire to Eat	15.43 (16.03)	12.67 (13.07)
	Thirstiness	28.04 (24.02)	28.86 (23.46)

Food Intake

Mean food intake (kcal), during the bogus taste test, is presented in Figure 3. In line with our hypothesis, OW/OB participants consumed significantly more calories of highly palatable food items than did NW participants, $F(1, 90) = 4.96$, $p = 0.03$. Total food intake (kcal) was only marginally significantly different between NW and OW/OB participants, $F(1, 90) = 3.75$, $p = 0.06$. No group difference was observed concerning the energy intake from fruits and vegetables, $F(1, 90) = 1.46$, $p = 0.23$.

However, when gender and dietary restraint, as assessed via the DEBQ, were entered as a covariate in the model, no group differences were observed in total energy intake, $F(1, 92) = 1.26, p = 0.27$, energy intake of highly palatable food items, $F(1, 92) = 2.28, p = 0.14$ and energy intake from fruits and vegetables, $F(1, 92) = 2.78, p = 0.10$.

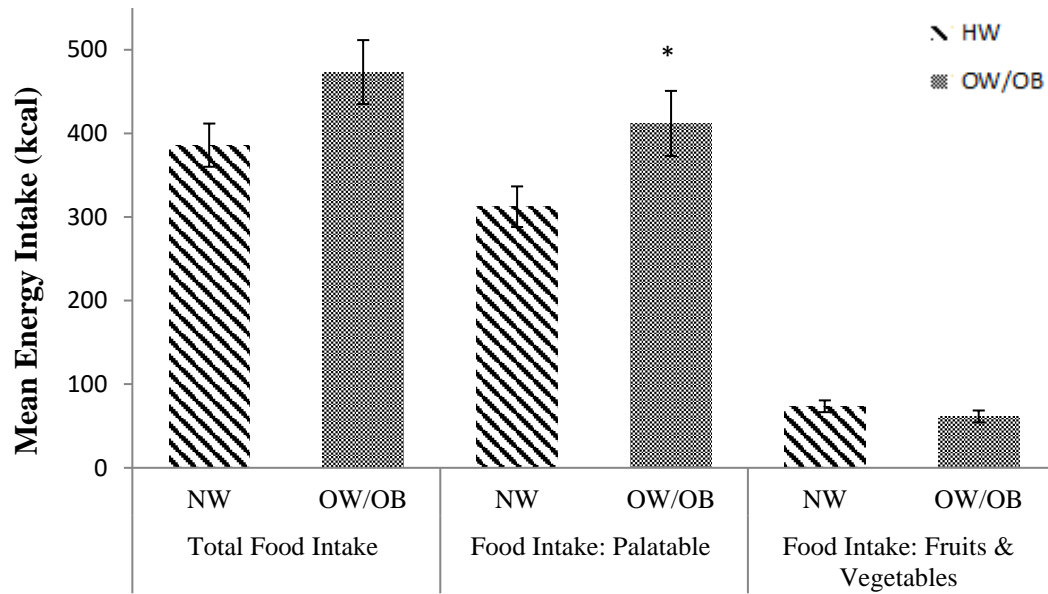


Figure 3. Mean food intake (kcal) by group. * $p < 0.05$

Liking ratings

Liking ratings of the offered food items by group are presented in Table 4. No effect of group was observed upon the liking ratings of the highly palatable food items, $F(4, 84) = 6.71, p = 0.61$; Wilk's $\Lambda = 0.97$, partial $\eta^2 = 0.03$. In addition, liking ratings of the highly palatable food items did not differ based upon participants gender $F(4, 84) = 1.18, p = 0.33$; Wilk's $\Lambda = 0.95$, partial $\eta^2 = 0.05$. There was no significant effect of group on the liking ratings of fruits and vegetables $F(4, 85) = 1.39, p = 0.25$; Wilk's $\Lambda = 0.94$, partial $\eta^2 = 0.06$ or a significant effect of gender $F(4, 85) = 0.96, p = 0.47$; Wilk's $\Lambda = 0.96$, partial $\eta^2 = 0.04$. Adding the dietary restraint, as assessed via the DEBQ, as a covariate in the models, had no effect.

Table 4*Mean Subjective Liking Ratings by Group*

Liking Ratings	Normal-weight (n = 49)		Overweight/Obese (n = 43)	
	Mean (SD)	Min-Max	Mean (SD)	Min-Max
Highly palatable food items				
Chocolate	68.80 (16.62)	7.70-96.70	68.36 (19.78)	23.70-100.00
Chocolate cookies	68.92 (17.86)	23.00-100.00	60.68 (26.60)	8.70-100.00
Crisps	53.88 (23.02)	9.70-97.00	58.70 (20.47)	8.30-100.00
Salted biscuits	52.60 (22.39)	7.70-98.00	59.52 (20.49)	11.30-95.70
Fruits & Vegetables				
Nectarine	71.38 (17.20)	14.00-99.00	61.77 (19.87)	18.70-94.70
Melon	71.41 (21.79)	12.30-100.00	63.94 (22.52)	6.70-100.00
Cherry tomatoes	50.59 (22.50)	0.00-92.30	52.53 (19.28)	6.30-87.30
Cucumber	66.70 (22.10)	9.30-100.00	58.62 (25.63)	1.70-96.70

Task Performance

In both the priming and working memory task, the accuracy for the search task was high. In the priming task, responses on catch trials were withheld as instructed with an average of 92% correct; in the working memory task, responses to the memory task were correct in 87% of all cases. There was no evidence of a speed–accuracy trade off.

Mean RTs (in milliseconds) to food and non-food cues for valid, invalid, and neutral trials, for both the priming and the WM tasks, are presented in Figure 3 for NW and OW/OB participants separately. RTs were slower in the WM than the priming task ($F(1, 90) = 119.24$;

$p < 0.001$, $\eta^2 = 0.57$) suggesting that the participants were performing these tasks differently. There was a main effect of validity ($F(2, 180) = 254.03$; $p < 0.001$, $\eta^2 = 0.74$), whereby RTs were faster for valid trials than the neutral and invalid trials, and were faster for neutral compared to invalid trials (all $p < 0.01$). There was also a main effect of cue ($F(1, 90) = 39.95$; $p < 0.001$, $\eta^2 = 0.31$), whereby RTs for food cues were faster than RTs for non-food cues. There was a significant two-way interaction between task and cue ($F(1, 90) = 8.77$; $p = 0.004$, $\eta^2 = 0.09$), RTs were shorter for food cues in both the priming ($p = 0.01$) and WM task ($p < 0.001$), however the difference was smaller in the priming task. In addition, there was a significant two-way interaction between task and validity ($F(2, 180) = 27.43$; $p < 0.01$, $\eta^2 = 0.23$); RTs were shorter for valid trials compared to invalid trials ($p < 0.001$), as well as the neutral trials ($p < 0.001$) in the WM task. A similar pattern was observed in the priming task (all $p_s < 0.001$), however, the difference between valid and neutral trials, and between valid and invalid trials was smaller. The two-way interaction between validity and cue ($F(2, 180) = 20.19$; $p < 0.01$, $\eta^2 = 0.18$) was also significant; RTs were shorter following food cues compared to non-food cues in the valid ($p < 0.001$) and neutral trials ($p < 0.001$), while no differences were observed in the invalid trials ($p = 0.67$).

To further explore the validity by cue interaction, RT for valid trials was subtracted from RT for invalid trials. Increased values on this score are suggestive of longer RT for invalid trials compared to valid trials, which translates to increased effort to disengage from the cued item when it was a distractor. Paired sample t-tests showed that when the cue was a food item the biasing effect was greater than when it was a non-food item for both the priming ($t(91) = 2.95$; $p < 0.01$) and the WM task ($t(91) = 4.81$; $p < 0.001$). However, the biasing effect was significantly greater in the WM task than the priming task, both for the food cues ($t(91) = 5.25$; $p < 0.01$) and the non-food cues ($t(91) = 4.91$; $p < 0.01$), suggesting that holding

a cue in WM has a significantly greater effect in biasing attention to visually similar stimuli even if it is irrelevant to the subsequent search task than primed with a cue (e.g. by the sight of the cue). One way ANOVA with group as a factor revealed that the biasing effect for non-food cues did not differ between NW and OW/OB participants, for both the priming ($F(1,90) = 0.21, p = 0.65$) and the WM task ($F(1,90) = 0.76, p = 0.39$). However, OW/OB participants showed a significantly greater biasing effect for the food cues in the WM task ($F(1, 90) = 4.96; p = 0.03$), while no difference was observed in the priming task ($F(1, 90) = 1.56; p = 0.22$), suggesting that holding food cues in WM, leads to greater attentional bias when the food cue is present, even if this is irrelevant to the task, in OW/OB but not in NW participants (see Figure 4).

The three-way interaction between task, validity, and cue ($F(2, 180) = 1.63; p = 0.20, \eta^2 = 0.02$) was not significant. The correction for gender did not change the three-way interaction ($F(2, 176) = 1.42; p = 0.24, \eta^2 = 0.02$). There was no significant effect of group ($F(1, 90) = 1.97; p = 0.16, \eta^2 = 0.02$), indicating that mean RTs from NW and OW/OB participants, were similar. There was no significant interaction between task and group ($F(1, 90) = 1.48; p = 0.23, \eta^2 = 0.02$), cue and group ($F(1, 90) = 0.56; p = 0.46, \eta^2 = 0.006$), validity and group ($F(2, 180) = 2.51; p = 0.08, \eta^2 = 0.03$), task, cue and group ($F(1,90) = 0.52; p = 0.47, \eta^2 = 0.006$), task, validity and group ($F(2,180) = 0.57; p = 0.57, \eta^2 = 0.01$), cue, validity and group ($F(2,180) = 1.81; p = 0.17, \eta^2 = 0.02$). The four-way interaction between task, validity, cue and group was also not significant ($F(2,180) = 2.21; p = 0.81, \eta^2 = 0.002$). Similarly, the correction for gender did not change the four-way interaction ($F(2, 176) = 0.29; p = 0.75, \eta^2 = 0.003$).

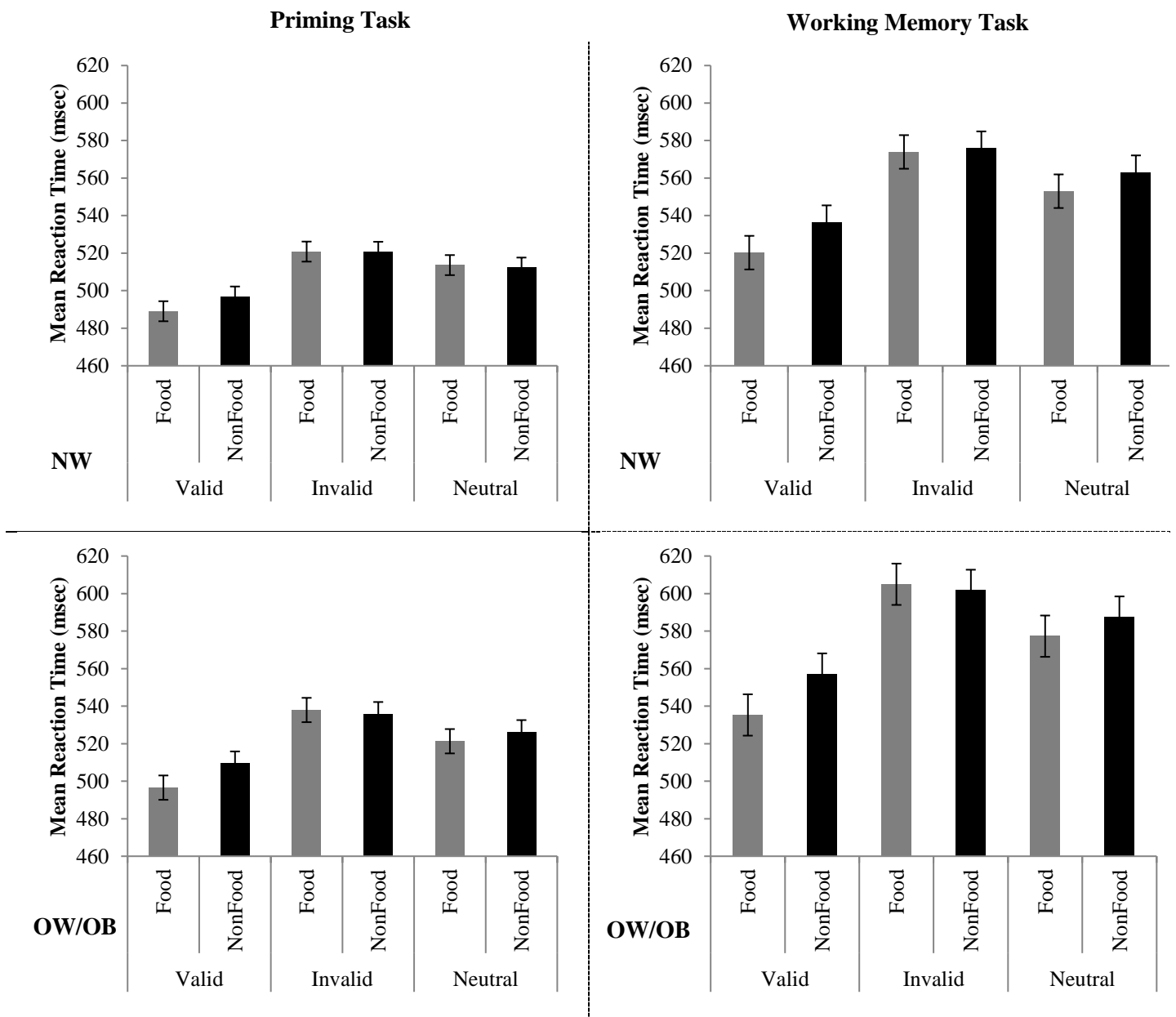


Figure 4. Mean reaction times (in milliseconds) to food, and non-food cues for Valid, Invalid, and Neutral trials, for the priming and working memory task and by group. Values are means \pm SEM.

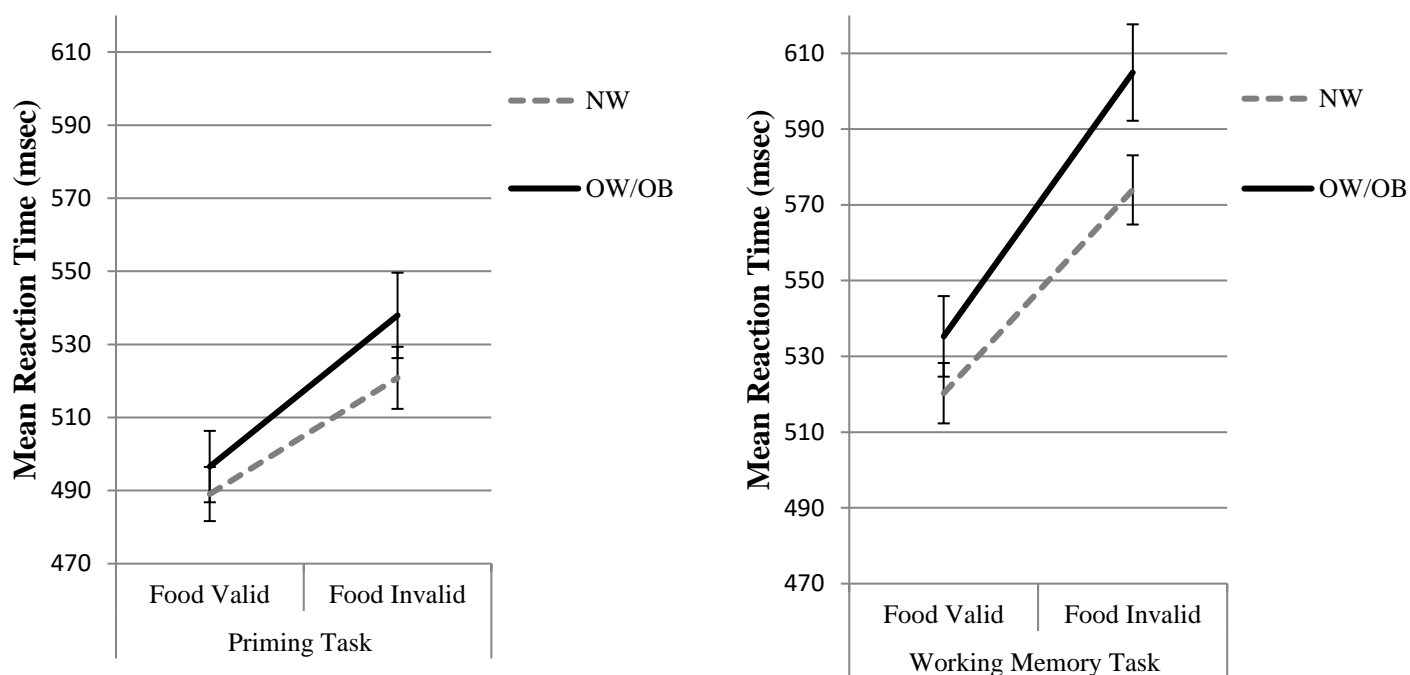


Figure 5. Mean reaction times (in milliseconds) to food cues for Valid and Invalid trials, for the priming and working memory task and by group. Although the HW participants were slower at invalid trials compared to valid trials, this validity effect was exacerbated for the OW/OB participants in the WM task.

Predictors of Food Intake

A regression analysis was conducted in order to examine whether the measure of effort to disengage from a cued item, when the item was held in WM (top-down guidance of attention) could predict food intake of palatable food items, as assessed in the bogus taste test, while taking into account additional possible predictors of food intake. Age, gender, subjective desire to eat ratings as assessed after the offered meal, mean liking ratings of the offered food items, levels of current physical activity, body fat (Kgs), lean weight (Kgs), trait disinhibition (as assessed via the TFEQ) and dietary restraint (as assessed via DEBQ), drive for consumption of palatable foods (e.g. PFS), impulsivity and the biasing effect of food when hold in WM were concurrently entered as predictors in the model. Trait disinhibition

correlates significantly with hunger (TFEQ-Hunger; $r = 0.49$, $p < 0.001$), emotional (BEBQ-Emotional, $r = 0.59$, $p < 0.001$) and external eating (BEBQ-External, $r = 0.58$, $p < 0.001$). In addition, dietary restraint as assessed via the DEBQ correlates significantly with the TFEQ-Restraint ($r = 0.81$, $p < 0.001$). Therefore, to avoid multicollinearity these variables were not entered as predictors in the model. Food intake from palatable food items (kcal) was the dependent variable. This model accounted for 40% of the variance in intake of palatable food items. Subjective ratings of desire to eat, as assessed after the offered meal, physical activity, and impulsivity were the only significant predictors of intake of palatable food items (see Table 5). There was also a trend for body fat to predict intake of palatable food items ($p = 0.06$).

Table 5*Multiple Regression Model Predicting Food Intake from Highly Palatable Food Items*

Predictor	Food Intake: Palatable		
	B	SE B	β
Age	-1.68	2.06	-0.08
Gender	17.12	91.52	0.03
VAS Desire to Eat (After offered Meal)	2.61	0.99	0.26*
VAS Liking	1.75	1.28	1.23
Physical Activity (MET-minutes/week)	0.02	0.01	0.25**
Body Fat (Kgs)	3.93	2.04	0.22
Lean Weight (Kgs)	3.51	3.38	0.19
TFEQ-Disinhibition	10.55	7.46	0.16
DEBQ-Restraint	-12.05	25.27	-0.05
PFS-Total	-50.17	30.60	-0.18
BIS-Total	5.82	2.17	0.25**
Biasing effect of food when hold in WM	1.02	0.65	0.16
ANOVA	F (12,79) = 4.42***		
R²	0.40		

*p< 0.05; **p<0.01

One-year Follow-up

Participants. One year later 57 participants returned back to the research unit for the follow up part of the study and an additional 13 participants provided self-report data of current body weight. Thus, in total, body weight data were available for 70 participants (76.1% of the initial sample); 34 OW/OB and 36 HW participants. In total, they were 52 females (74.3%) and 18 males (25.7%).

BMI change. A change variable was computed by subtracting participants' BMI at phase 1 from their BMI at phase 2. Higher scores indicated a greater increase in BMI. Results of a paired-sample t-test revealed only a trend towards significant increase in BMI, $t(69) = 1.71$, $p = 0.09$ (Mean BMI Phase 1 = 26.05, s.d. = 6.06; Mean BMI Phase 2 = 26.27, s.d. = 6.43).

Predictors of BMI change. A regression analysis was conducted in order to examine whether the biasing effect for the food cues in the WM task could predict BMI change over a one-year period, while taking into account additional possible predictors of BMI change. Age, gender, levels of physical activity, body fat (Kgs), lean weight (Kgs), trait disinhibition and dietary restraint (as assessed via DEBQ), drive for consumption of palatable foods (e.g. PFS), impulsivity and the biasing effect of food when held in WM, as assessed at phase 1 of the study were concurrently entered as predictors in the model. BMI change was the dependent variable. This model accounted for 35% of the variance in BMI change over a one-year period. Gender, lean weight and the biasing effect of food when held in WM were the only significant predictors of BMI change (see Table 6).

Table 6*Multiple Regression Model Predicting BMI Change Over a One-Year Period*

Predictor	BMI Change: 1 Year Follow-up		
	B	SE B	β
Age	0.02	0.01	0.15
Gender	2.14	0.59	0.88**
Physical Activity (MET-minutes/week)	5.765E-5	0.00	0.14
Body Fat (Kgs)	-0.00	0.01	-0.04
Lean Weight (Kgs)	0.06	0.02	0.68**
TFEQ-Disinhibition	0.07	0.04	0.21
DEBQ-Restraint	-0.09	0.16	-0.07
PFS-Total	-0.15	0.19	-0.11
BIS-Total	-0.02	0.01	-0.16
Biasing effect of food when hold in WM	0.01	0.00	0.26*
ANOVA	F (10,59) = 3.16**		
R²	0.35		

*p< 0.05; **p<0.01

2.5 Discussion

The aim of this study was to investigate whether attentional biases towards food cues distinguish overweight/obese from normal-weight participants, and to assess whether attentional biases for food cues can predict subsequent food consumption, as assessed in a laboratory setting, and weight gain over a one-year period. Overweight/obese participants showed greater attentional biases toward food cues than did normal-weight participants, but only in the working memory task, that is when they were holding food related information in working memory. During the priming task, food cues had a greater biasing effect than non-food cues as assessed during the subsequent selection task, but no difference was observed

between normal-weight and overweight/obese participants. In addition, during the taste test, overweight/obese participants consumed more of the offered food items than normal-weight participants, but only for the highly palatable food items; energy intake from fruits and vegetables did not differ across these groups. Attentional biases towards food cues, when food related information was held in working memory, was found to predict weight gain (BMI change) over a one-year period, after taking into account important possible predictors of BMI change.

To measure attentional biases for food cues in this study, we used a paradigm that captures both automatic (bottom-up priming condition) and top-down guidance of attention (the top-down working memory condition). Both overweight/obese and normal-weight subjects demonstrated an attentional bias for food cues in the bottom-up priming condition, suggesting that food cues bias attention more than non-food cues due to greater salience (Castellanos et al., 2009). However, in the working memory condition, that is when participants were asked to hold food-related information in working memory, overweight/obese compared to normal-weight participants showed greater attentional biases for food cues, as they were much slower to detect the target in the invalid trials compared to valid trials than healthy-weight subjects, indicating that in a condition where food cues are available and match representations in working memory, overweight/obese individuals find it much harder to disengage from those cues even if they are irrelevant to current goals (e.g. target selection). These findings are suggestive of a novel mechanism that underlies attention allocation to food stimuli in overweight/obesity through working memory even under conditions of satiety, as in the current study. Castellanos et al. (2009) also reported attentional biases for food cues in obese individuals when sated. Cox, Pothos, and Hosier (2007) have proposed an analogous hypothesis in a motivational framework, according to which

preoccupation with an issue, such as alcohol consumption, would lead to cognitive biases for related information. However, to the best of our knowledge, this is the first study to directly assess guidance of attention via working memory, under conditions which mimic situations of food preoccupation that many people with obesity report (Israel et al., 1985; Lynch, Eppers, & Sherrodd, 2004).

A further aim of this study was to investigate whether attentional biases for food cues can predict eating behaviour, as assessed in a laboratory setting via a bogus taste test, and actual weight gain over a one-year period. We observed a different eating pattern for the overweight/obese group compared to the normal-weight group; overweight/obese individuals consumed more energy from highly palatable foods than normal-weight individuals, while no difference was observed in the consumption of fruits and vegetables. Food intake data demonstrated a difference in consumption of highly palatable food items of approximately 100 calories. Werthmann et al. (2011), in accordance with our findings also reported greater snack consumption in overweight/obese individuals than normal-weight controls. However, the mean reported difference was smaller (≈ 50 calories) than that observed in our study; the recruitment of a more overweight/obese sample in the current study (mean BMI: 30.46 vs. 28.03 kg/m²) might explain this difference. Attentional biases for food cues, did not predict food intake of highly palatable food items. Werthmann et al. (2011) and Nijs et al. (2010) also found no association between attentional biases to food and snack food intake, suggesting that the relationship between attentional biases and food intake might be complex. However, our model of food intake, as assessed during a bogus taste test explained 40% of the variance in food intake of highly palatable food items.

Subjective ratings of desire to eat, as assessed after the offered meal, levels of current physical activity, and impulsivity were the only significant predictors of food intake from

palatable food items. There was also a trend for body fat levels to predict food intake from palatable food items, which is consistent with our finding that overweight/obese individuals consumed more of the highly palatable food items during the bogus taste test than the healthy-weight individuals. Impulsivity, has been suggested to play a critical role in the aetiology and maintenance of obesity (Guerrieri, Nederkoorn, & Jansen, 2008). For example, higher impulsivity has been found to correlate with food intake in laboratory studies (Guerrieri, Nederkoorn, & Jansen, 2007), fast food consumption (Churchill & Jessop, 2011; Garza, Ding, Owensby, & Zizza, 2016) and unsuccessful dieting (Meule, Papies, & Kübler, 2012).

Similarly, in food-related decision tasks, more impulsive individuals were found to show more unhealthy eating behaviours compared to individuals with lower impulsivity (Jasinska et al., 2012; Sengupta & Zhou, 2007). Physical activity levels were also found to positively predict food consumption of highly palatable food items, that is participants who reported greater levels of current physical activity consumed more energy from highly palatable food items. This finding is not unexpected given that subjects who exercise frequently have increased energy requirements (Campbell, Crim, Young, & Evans, 1994). Desire to eat, as assessed subjectively via the VAS, was also a significant predictor variable in our model, and increased scores of reported desire to eat positively predicted energy intake from highly palatable food items. Barkeling, Rossner, and Sjoberg (1995) tested the predictive validity of subjective motivation to eat ("desire to eat", "hunger", "fullness" and "prospective consumption") on VAS and found that only the variables "desire to eat" and "prospective consumption" predicted subsequent food intake. In our sample, only subjective ratings of desire to eat but not hunger were significantly related to consumption of highly palatable food items. Existing models of food intake, as assessed in a laboratory setting, most

of often include baseline hunger as a potential predictor of food intake (Robinson et al., 2017), and our findings suggest that desire to eat may be a better predictor of food intake, especially under conditions of satiety as in the present study.

Attentional biases towards food cues, when food related information was held in working memory, was found to predict weight gain (BMI change) over a one-year period, after taking into account important possible predictors of BMI change. To the best of our knowledge, only two other studies have investigated the predictive value of attentional biases in weight gain (Calitri et al., 2010; Yokum et al., 2011), and only one used pictorial food stimuli (Yokum et al., 2011), as in the current study. In line with previous research, our findings are supportive of a positive association between attentional biases for food cues and weight gain. For first time however, we provide evidence that subjects who show enhanced attentional biases for food cues when holding food-related information in working memory, are at increased risk for future weight gain, highlighting the importance of top-down mechanisms in the regulation of body weight.

In addition, compared to Calitri et al. (2010) our model of BMI change explained a significantly higher proportion of variance in BMI change (35% vs. 18%), suggesting that the studied factors should be included in future models aimed to investigate predictors of weight gain. Our results demonstrated that gender and lean weight, but not body fat mass, were also significant predictors of BMI change over a one-year period. Thus, female participants gained more weight than male participants and subjects with greater lean weight at baseline also gained more weight at one-year follow-up. However, gender was not equally distributed in our sample, and only 25.7% of participants at one-year follow-up were males. Therefore, our finding that females are at increased risk for weight-gain should be interpreted with caution, and future studies with more equally distributed samples of males and females are warranted.

Interestingly, we found that lean weight but not body fat mass levels significantly predicted weight gain over a one-year period. Accumulating evidence suggests that fat-free mass plays a major role in appetite regulation, stimulating food intake and our data contribute to this evidence base (Hopkins & Blundell, 2016; MacLean, Blundell, Mennella, & Batterham, 2017).

In conclusion, the current study provides evidence for individual differences in attentional biases for food cues in overweight/obese versus normal-weight participants. Overweight/obese subjects showed an enhanced bias toward food cues when food-related information was held in working memory, indicating that holding food-related information in working memory, can be particularly effective in guiding attention to similar stimuli even if it is irrelevant to current goals, providing for the first-time evidence for a novel mechanism underlying attentional biases in overweight/obese individuals. In addition, the longitudinal association that was observed between the biasing effect of food, when relevant information was held in working memory and weight gain over a one-year period is suggestive of a causal interplay between these two variables. An important question for future research is to assess whether the efficacy of treatments for obesity (e.g. behavioural or pharmacological interventions) is dependent upon the extent to which they influence top-down guidance of attention from working memory, resulting in reduced attentional bias for food cues.

Chapter 3:

Systematic Literature Review

Kaisari, P., Dourish, C. T., & Higgs, S. (2017). Attention Deficit Hyperactivity Disorder (ADHD) and disordered eating behaviour: A systematic review and a framework for future research. *Clinical Psychology Review*, 53, 109-121².

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Review

Attention Deficit Hyperactivity Disorder (ADHD) and disordered eating behaviour: A systematic review and a framework for future research



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HIGHLIGHTS

- Symptoms of Attention Deficit Hyperactivity Disorder (ADHD) are associated with disordered eating.
- Impulsivity symptoms of ADHD are positively associated with bulimic behaviours.
- A causal effect of ADHD on disordered eating cannot be inferred.
- Mechanistic studies on the link between ADHD and disordered eating are required.

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ABSTRACT

Preliminary findings suggest that Attention Deficit Hyperactivity Disorder (ADHD) may be associated with disordered eating behaviour, but whether there is sufficient evidence to suggest an association between ADHD and specific types of disordered eating behaviour is unclear. Furthermore, it is uncertain whether specific features associated with ADHD are differentially associated with disordered eating behaviour. A systematic review of seventy-five studies was conducted to evaluate the potential association between ADHD symptomatology and disordered eating behaviour and to provide an estimate of the strength of evidence for any association. Overall, a moderate strength of evidence exists for a positive association between ADHD and disordered eating and with specific types of disordered-eating behaviour, in particular, overeating behaviour. There is consistent evidence that impulsivity symptoms of ADHD are positively associated with overeating and bulimia nervosa and more limited evidence for an association between hyperactivity symptoms and restrictive eating in males but not females. Further research is required to assess the potential direction of the relationship between ADHD and disordered eating, the underlying mechanisms and the role of specific ADHD symptoms in the development and/or maintenance of disordered eating behaviour. We propose a framework that could be used to guide the design of future studies.

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1. Introduction

Attention Deficit Hyperactivity Disorder (ADHD) is one of the most common neurodevelopmental disorders of childhood (Polanczyk, Willcutt, Salum, Kieling, & Rohde, 2014) and has a worldwide prevalence of 5% in school-age children (Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007). The symptoms of ADHD persist in adults in up to 65% of cases (Faraone, Biederman, & Mick, 2006) and the prevalence of ADHD in adults is estimated at 2.5% (Simon, Czobor, Bálint, Mészáros, & Bitter, 2009). Despite the high prevalence of the disorder, fewer than 20% of adults with ADHD are diagnosed or treated (Ginsberg, Quintero, Anand, Casillas, & Upadhyaya, 2014). Moreover, up to 90% of adults with ADHD have comorbid psychiatric disorders (Nutt et al., 2007), which may obscure the symptoms of ADHD. Depression and other mood disorders, anxiety, personality disorders and substance use disorders (SUDs), in addition to oppositional defiant disorder, sleep problems and learning disabilities are often comorbid with ADHD (Corbisiero, Stieglitz, Retz, & Rosler, 2013; Gillberg et al., 2004; Lin, Yang, & Gau, 2015; Miller, Nigg, & Faraone, 2007; Sobanski et al., 2007).

It has also been reported that there is an association between ADHD and eating disorders (EDs) (Bleck & DeBate, 2013; Mikami et al., 2010; Mikami, Hinshaw, Patterson, & Lee, 2008). A scoping search retrieved four reviews relevant to the relationship between ADHD and eating disorders (Cortese, Bernardina, & Mouren, 2007; Curtin, Pagoto, & Mick, 2013; Nazar et al., 2008; Ptacek et al., 2016). Three of these reviews were narrative rather than systematic reviews (Cortese et al., 2007; Nazar et al., 2008; Ptacek et al., 2016). The only published systematic review focussed on individuals aged 12–21 years (Curtin et al., 2013) and thus it is unclear whether disordered eating behaviours are also present in younger children with ADHD. Furthermore, the onset of some EDs such as Binge Eating Disorder (BED) is usually later in adult life (Fairburn & Harrison, 2003), between the ages of 30 and 40 years, highlighting the importance of assessing older individuals. Our aim is to address this gap in the literature by conducting the first systematic review of the association between ADHD and disordered eating in both children and adults.

The present review will also extend knowledge of the relationship between ADHD and disordered eating by including studies that adopt a broader sampling frame than previous reviews. There is evidence that patterns of eating behaviour span a spectrum from extreme over-control of eating to loss of control and binge eating (Lowe et al., 1996; Wierenga et al., 2014), and that disordered eating patterns which do not meet clinical criteria are, nevertheless, often associated with psychopathology, and may eventually develop into a diagnosed eating disorder (Tanofsky-Kraff, Engel, Yanovski, Pine, & Nelson, 2013). Hence, greater insight into the relationship between ADHD and EDs will be

gained from studying the full range of variation of eating traits and behaviours, including restrained eating, food craving and loss of control over eating (Herman & Mack, 1975; Nammi, Koka, Chinnala, & Boini, 2004; van Strien, Herman, & Verheijden, 2012; Zeeck, Stelzer, Linster, Joos, & Hartmann, 2011). This is especially relevant for young children and adolescents, for whom diagnostic criteria for EDs may not be applicable (Bravender et al., 2007; Bravender et al., 2010).

We also aim to identify studies that have investigated whether specific features of ADHD are differentially associated with specific types of disordered eating. Recent research has emphasised the role of cognitive processes in the control of eating behaviour (Higgs, 2016). Studying the relationship between individual variations in attention and cognitive control and disordered eating in ADHD provides an opportunity to identify core processes that cut across diagnostic categories and could be targeted by therapeutic interventions.

The first aim of this paper was to systematically review the literature for an association between ADHD and disordered eating. The following four questions were addressed:

- (1) Is there an association between ADHD symptoms and disordered eating behaviour?
- (2) Are specific features of ADHD differentially associated with specific types of disordered eating behaviour?
- (3) Are there factors that affect the direction and/or strength of any relationship between ADHD symptoms and disordered eating (moderators)?
- (4) Which factors could explain the relationship between ADHD symptoms and disordered eating behaviour (mediators)?

A second aim was to use our evaluation of the current evidence base to make suggestions for future research. The proposed framework could also be applied to the study of other psychiatric disorders, such as schizophrenia and mood disorders, that are associated with disordered eating.

2. Materials and methods

2.1. Literature search strategy

Original research studies examining the association between ADHD and disordered eating were selected through a literature search in 3 electronic databases: PubMed, Ovid Databases (MEDLINE, PsycINFO, EMBASE + EMBASE CLASSIC) and Web of Science-Core Collection (from 1900). The literature search was performed during May 2016 by a single investigator (PK). Searches included a combination of key words relevant to disordered eating behaviour and ADHD. For the full search strategy used, see Supplemental material. Search limiters

included human subjects and English language. These electronic searches were supplemented by a manual search of reference sections in articles identified by the electronic search and other relevant sources. The search process was guided by the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.

2.2. Study selection

All original, peer-reviewed studies excluding case series/case studies and drug studies were considered suitable for inclusion. Case series/case studies were excluded as the level of evidence provided by this type of study is recognised as low (<http://www.cebm.net/ocebm-levels-of-evidence/>). The research evidence relating to the effect of ADHD medication on appetite and disordered eating behaviour is beyond the aim of this review and therefore drug studies were not considered for inclusion. There was no exclusion based on the age of the study participants and the selected studies included children, adolescents and/or adults with either: (1) a categorical diagnosis of ADHD according to the DSM (III, III-R, IV, IV-TR, V) or (2) or Hyperkinetic Disorder (HKD) as per the ICD-10 or previous ICD versions; or (3) for adults, a positive answer to the question: "Did your doctor ever tell you that you have ADHD?" or (4) a diagnosis of ADHD recorded in medical files/registries; or (5) a definition of ADHD using a symptoms threshold measure on a validated ADHD rating scale; or (6) ADHD symptoms measured dimensionally.

2.3. Data extraction

Data extraction was performed using standardized forms created for the review and each article was evaluated by two reviewers. The second reviewer (SH) confirmed the first reviewer's (PK) data extraction for completeness and accuracy. The following data were extracted: age range, sample size, sex distribution, study design, source population (e.g., psychiatrically referred or population-based data), methods of assessing ADHD and disordered eating, findings on the association between ADHD and disordered eating, findings on the association between specific symptoms of ADHD and disordered eating, covariates and moderator and mediators. The fully adjusted measure of association was reported if available.

2.4. Data synthesis

Data were organised according to the type of disordered eating behaviour. Where studies presented results concerning different types of disordered eating behaviour, data were included in all relevant sections. If only a limited number of studies (≤ 3 studies) presented data for a specific type of disordered eating behaviour, data were not narratively synthesised, as no conclusions could be drawn with confidence at this stage. These included data concerning food addiction (FA) (Davis et al., 2011), meal skipping (Pagoto et al., 2010; Ptacek et al., 2014), meal/eating frequency (Blomqvist, Ahadi, Fernell, Ek, & Dahllof, 2011; Blomqvist, Holmberg, Fernell, Ek, & Dahllof, 2007; Ptacek et al., 2014), Night Eating Syndrome (NES) and/or waking up at night to eat (Docet, Larranaga, Perez Mendez, & Garcia-Mayor, 2012; Nicolau et al., 2014; Runfola, Allison, Hardy, Lock, & Peebles, 2014), snacking (Docet et al., 2012; Ebenegger et al., 2012) and selective/picky eating (Machado, Dias, Lima, Campos, & Gonçalves, 2016; Zucker et al., 2015). Due to the degree of heterogeneity among study designs, particularly with respect to the population characteristics, the assessment methods of EDs and/or eating pathology/disordered eating and outcome measures, a meta-analysis was not conducted.

2.5. Quality and strength of evidence assessment

Critical appraisal checklists were used to evaluate the quality of the studies. Checklists were adapted from the Newcastle–Ottawa Quality Assessment Scale (Normand et al., 2005; Wells et al., 2000) and critical

appraisal articles (Grimes & Schulz, 2002; Gurwitz et al., 2005; Mamdani et al., 2005; Normand et al., 2005). The Newcastle–Ottawa Quality Assessment Scale has established content validity and inter-rater reliability (Wells et al., 2000). Items reviewed included the representativeness of sampling procedure, the response rate, the validity of the measurement methods and control for important confounders. A response rate of 60% or above was considered adequate. This cut-off has been used in previous systematic reviews of observational studies (Garipey, Nitka, & Schmitz, 2010; Wong, Cheung, & Hart, 2008). Reporting on the methodological aspects of the studies rather than relying on a numerical score for quality is considered more appropriate for systematic reviews and meta-analyses (Juni, Witschi, Bloch, & Egger, 1999). Therefore, we rated individual components of the checklist (criteria met, criteria not met, not reported) and provided an overall rating for the quality of the study (low, moderate or high). Quality assessment of studies was conducted independently by two investigators (PK and SH). Any disagreements were resolved by discussion or, if necessary, through a third investigator (CD).

Four categories were used to grade the evidence: (1) "high" grade (indicating high confidence that the evidence reflects a true association, and further research is unlikely to change the confidence level in the estimate of the association); (2) "moderate" grade (indicating moderate confidence that the evidence reflects a true association, and further research may change the confidence level in the estimate of the association and could change the estimate); (3) "low" grade (indicating low confidence that the evidence reflects a true association, and further research is likely to change the confidence level in the estimate of the association and the estimate); and (4) "insufficient" grade (indicating evidence is unavailable or limited, and a conclusion could not be drawn based on the available data).

The body of evidence was considered as consistent in direction if $\geq 70\%$ of the studies had an effect in the same direction (e.g., showed significant association versus no association).

3. Results

3.1. Study selection

Fig. 1 illustrates the flowchart of study selection (Fig. 1; PRISMA flow chart). The key word search initially identified 6376 citations, 874 from PubMed, 1852 from Web of Science and 3650 from Ovid Databases. After initial screening based on the title and abstract of the article, 184 articles remained for full assessment of which 78 were excluded because they had not evaluated the relationship between ADHD and eating behaviour, 40 were review articles and 1 presented the same results as another report. Seven additional articles (Hudson, Hiripi, Pope, & Kessler, 2007; Levy, Fleming, & Klar, 2009; Mattos et al., 2004; Nazar et al., 2012; Neumark-Sztainer, Story, Resnick, Garwick, & Blum, 1995; Steadman & Knouse, 2014; Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011) identified through searching reference lists identified in the electronic search were also included. One article reported 4 studies that met the inclusion criteria (Surman, Randall, & Biederman, 2006). Thus, a total of 72 articles reporting on 75 suitable studies were reviewed. Due to the limited number of studies (≤ 3 studies) presenting data for a specific type of disordered eating behaviour, data from 8 studies were not analysed further (Blomqvist et al., 2011; Blomqvist et al., 2007; Davis et al., 2011; Ebenegger et al., 2012; Machado et al., 2016; Ptacek et al., 2014; Runfola et al., 2014; Zucker et al., 2015).

3.2. Characteristics of the included studies

Supplementary material summarizes the characteristics and main findings of the studies included. The total number of participants included in the current review was 115,418. Sample sizes of the 75 studies varied widely between 26 and 12,366. Most studies included both male and



PRISMA 2009 Flow Diagram

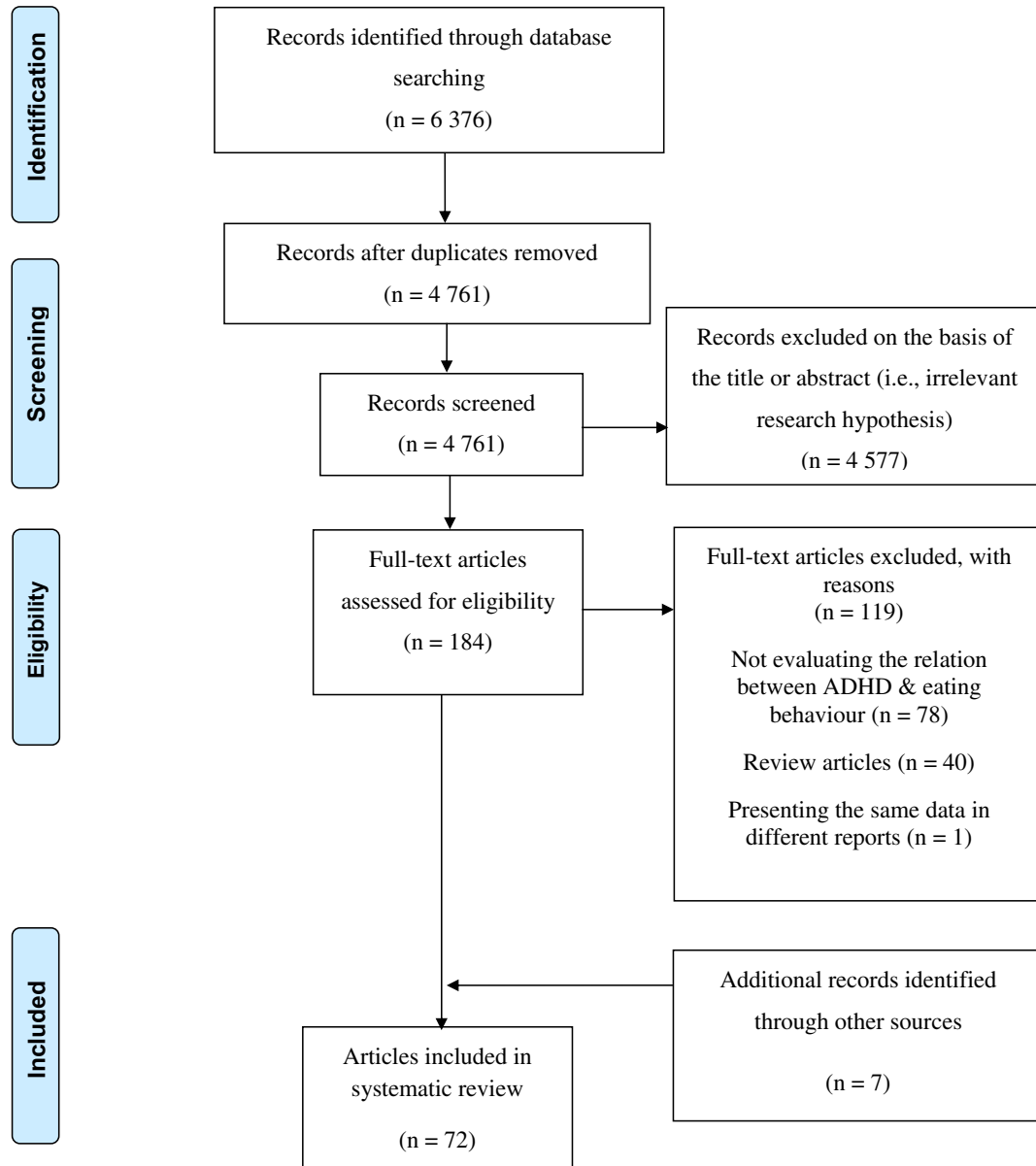


Fig. 1. Flow chart of study selection process.

female participants ($n = 57$) and of those studies the majority ($n = 34$) included more females than males. Fifteen studies included only females and 3 studies only males. The publication year ranged from 1995 to 2016 and 27 of the 75 studies were published within the last 3 years. Most studies were cross-sectional ($n = 37$), 11 studies were case-control, 6 were cohort studies, 5 were prospective, 7 were longitudinal or part of a longitudinal study, 2 were a secondary analysis of data from the National Longitudinal study of Adolescent Health, 3 were experimental studies, 3 studies were retrospective reviews and 1 was an epidemiological study. Thirty-five studies were conducted in Europe, thirty-seven in America and two in Asia. One study used data collected in the United States, Canada, Great Britain and the Netherlands. Thirty studies recruited children and adolescent participants (age range: 1.5–18 years), 35 studies recruited adults (age ≥ 18 years) and 10 studies

recruited children and/or adolescents and adults (minimum age: 6 years). Study participants were recruited from the general population ($n = 29$), university students ($n = 3$), children referred through community pediatric mental health clinics, medical clinics and flyers on public bulletin boards ($n = 1$), clinical populations with EDs ($n = 6$) and ADHD ($n = 15$), adults referred with ADHD and/or autism spectrum disorders (ASD) ($n = 1$), tic disorder specialty clinics in the United States, Canada, Great Britain and the Netherlands as from the Tourette Syndrome Association of the United States ($n = 1$), female offenders ($n = 1$), obese children who received, presented or were referred for weight-loss treatment ($n = 5$), obese women seeking non-surgical treatment for obesity ($n = 2$), obese patients attending the Nutrition section of a hospital ($n = 1$), obese patients who underwent or were candidates for bariatric surgery ($n = 6$), severely obese patients who

had been referred for the treatment of refractory obesity ($n = 1$) and obese individuals in community settings ($n = 3$). In 41 studies the association between ADHD and disordered eating was not the main focus of the study and estimates were usually displayed in a descriptive table.

3.3. ADHD and eating disorders (EDs)

Eleven studies reported data relevant to the association between ADHD and EDs, without providing any information about the type of ED (see Supplementary material). Two studies used a case-control design (Bijlenga et al., 2013; Sobanski et al., 2007), 1 study was a two-part longitudinal study with a one-year interval (Viborg, Wangby-Lundh, & Lundh, 2014), 1 study was a longitudinal study with a two-year follow-up (Rojo-Moreno et al., 2015), 1 study was a cohort study (Yoshimasu et al., 2012), 1 was an epidemiological study (Lewinsohn, Shankman, Gau, & Klein, 2004), 3 were cross-sectional studies (Hirschtritt et al., 2015; Karjalainen, Gillberg, Rastam, & Wentz, 2016; Stulz et al., 2013) and 2 studies were a secondary analysis of data from the US National Longitudinal Study of Adolescents Health (Bleck & DeBate, 2013; Bleck, DeBate, & Olivardia, 2015). All studies except two, which included only females (Stulz et al., 2013; Viborg et al., 2014) included both sexes. Overall, significant and positive associations between ADHD and EDs were found in 8 of the 11 studies.

3.4. ADHD and anorexia nervosa (AN)/restrictive eating

Nineteen studies reported data relevant to the association between ADHD and AN/Restrictive eating (see Supplementary material). Two studies were prospective (Biederman et al., 2007; Biederman et al., 2010), 10 studies were cross-sectional (Dempsey, Dyehouse, & Schafer, 2011; Grabarek & Cooper, 2008; Hudson et al., 2007; Karjalainen et al., 2016; Muller, Claes, Wilderjans, & de Zwaan, 2014; Pauli-Pott, Becker, Albayrak, Hebebrand, & Pott, 2013; Rosler, Retz, Yaqoobi, Burg, & Retz-Junginger, 2009; Slane, Burt, & Klump, 2010; Swanson et al., 2011; Wentz et al., 2005), 1 study was an ongoing longitudinal study (Rastam et al., 2013), 2 were part of longitudinal studies (Malmberg, Edbom, Wargelius, & Larsson, 2011; Yates, Lund, Johnson, Mitchell, & McKee, 2009), 2 studies were a secondary analysis of data from the US National Longitudinal Study of Adolescents Health (Bleck & DeBate, 2013; Bleck et al., 2015) and 2 studies were retrospective reviews (Blinder, Cumella, & Sanathara, 2006; Welch, Ghaderi, & Swenne, 2015). Fourteen studies included both sexes and 5 studies included only females.

Restrictive eating symptoms investigated across these studies included adoption of behaviours such as use of weight-loss pills, fasting or meal skipping, replacement of meals with food supplements or use of food supplements to reduce appetite in order to lose or maintain body weight, failure to gain sufficient weight for more than a year, fear of gaining weight or growing fat, fear of becoming obese, drive for thinness and restraint eating. Overall, 9 of 19 studies reported significant and positive associations between ADHD and AN/Restrictive eating and for 3 of these studies the associations were significant only for males (Grabarek & Cooper, 2008; Rastam et al., 2013; Welch et al., 2015). Three studies found significant associations only between specific symptoms of ADHD (hyperactivity and hyperactivity/impulsivity) and restrictive eating behaviour (Grabarek & Cooper, 2008; Rastam et al., 2013; Slane et al., 2010).

Population studies with large sample sizes (ranging from 5000 to 12,262 participants) in general reported a non-significant association between ADHD and AN/Restrictive eating symptoms (Bleck & DeBate, 2013; Hudson et al., 2007; Swanson et al., 2011). However, the failure to detect an association in these studies may be due to methodological issues rather than representing a true null result. Specifically, analyses of associations in some studies may have been underpowered owing to the relatively small numbers of participants with AN (Hudson et al.,

2007; Swanson et al., 2011) and to the measure used to assess restrictive eating pathology (Bleck & DeBate, 2013).

Overall, the studies that found significant associations were characterised as low quality, with only two studies rated as moderate quality (Bleck et al., 2015; Slane et al., 2010). The most important methodological limitations were small sample sizes (Grabarek & Cooper, 2008; Rosler et al., 2009; Welch et al., 2015; Wentz et al., 2005), study of a forensic population with a high prevalence of Axis I disorders (Rosler et al., 2009), use of non-validated tools to assess eating problems (Bleck et al., 2015; Rastam et al., 2013), absence of a control group (Grabarek & Cooper, 2008; Karjalainen et al., 2016; Malmberg et al., 2011; Rastam et al., 2013; Slane et al., 2010; Welch et al., 2015; Wentz et al., 2005) and lack of control for confounding variables (Grabarek & Cooper, 2008; Malmberg et al., 2011; Rastam et al., 2013; Slane et al., 2010; Welch et al., 2015; Wentz et al., 2005).

3.5. ADHD and bulimia nervosa (BN)

Twenty-one studies evaluated the association between ADHD and BN and/or BN symptoms (see Supplementary material). Five studies were prospective studies (Biederman et al., 2007; Biederman et al., 2010; Hinshaw et al., 2012; Mikami et al., 2010; Mikami et al., 2008), 5 studies used a case-control design (Seitz et al., 2013; Surman et al., 2006), 8 studies were cross-sectional studies (Cortese, Isnard, et al., 2007; De Zwaan et al., 2011; Grabarek & Cooper, 2008; Hudson et al., 2007; Nazar et al., 2012; Neumark-Sztainer et al., 1995; Rosler et al., 2009; Swanson et al., 2011), 2 studies were a secondary analysis of data from the US National Longitudinal Study of Adolescents Health (Bleck & DeBate, 2013; Bleck et al., 2015) and 1 study was a retrospective chart review (Blinder et al., 2006). Eight studies included only females and 13 included both sexes. Overall, significant and positive associations between ADHD and BN/BN symptoms were found in 17 of the 21 studies.

Prospective evidence generally showed a significant positive association between ADHD and BN/BN symptoms (Biederman et al., 2007; Biederman et al., 2010). Only one prospective study did not find a significant association between ADHD and BN/BN symptoms. Hinshaw et al. (2012) in contrast with their previous findings (Mikami et al., 2008) found that girls with childhood-diagnosed ADHD did not differ significantly from controls in terms of BN symptoms at a 10-year follow-up. However, the power of the analysis might have been limited because participants lost in follow-up were poorer, had lower IQ scores and were more symptomatic than the retained sample and there was a large variation observed in continued service utilisation and medication use over the follow-up period.

Findings from case-control studies also pointed to a significant association between ADHD and BN in adult females (Seitz et al., 2013; Surman et al., 2006). Similarly, results from cross-sectional studies suggested a positive association between ADHD and BN/BN symptoms (Cortese, Isnard, et al., 2007; De Zwaan et al., 2011; Hudson et al., 2007; Nazar et al., 2012; Neumark-Sztainer et al., 1995; Rosler et al., 2009; Swanson et al., 2011).

3.6. ADHD and binge eating disorder (BED)

Twenty-seven studies reported data relevant to an association between ADHD and BED symptoms (see Supplementary material). Most studies were cross-sectional ($n = 17$) (Agranat-Meged et al., 2005; Alfnsson, Parling, & Ghaderi, 2012; Davis, Cohen, Davids, & Rabindranath, 2015; De Zwaan et al., 2011; Gruss, Mueller, Horbach, Martin, & de Zwaan, 2012; Hudson et al., 2007; Mattos et al., 2004; Muller et al., 2012; Muller et al., 2014; Nazar et al., 2012; Nazar et al., 2014; Pagoto et al., 2009; Pauli-Pott et al., 2013; Rosler et al., 2009; Slane et al., 2010; Steadman & Knouse, 2014; Swanson et al., 2011), 2 studies used a case-control design (Docet et al., 2012; Nicolau et al., 2014), 1 study used a case-double control design (Davis et al., 2009), 1

study was a retrospective chart review (Reinblatt et al., 2015), 1 study was longitudinal (Goldschmidt, Hipwell, Stepp, McTigue, & Keenan, 2015), 1 was part of a longitudinal clinical intervention study (Levy et al., 2009), 2 studies were a secondary analysis of data from the US National Longitudinal Study of Adolescents Health (Bleck & DeBate, 2013) and 2 studies were cohort studies (Khalife et al., 2014; Sonnevile et al., 2015). Four studies recruited females and the remaining 23 studies recruited both sexes. Overall, significant and positive associations between ADHD and BED/BED symptoms were found in 20 of the 27 studies.

Of the studies that showed significant associations, 3 studies found significant associations between specific symptoms of ADHD (hyperactivity, hyperactivity/inattention and hyperactivity/impulsivity) and binge eating (Goldschmidt et al., 2015; Slane et al., 2010; Sonnevile et al., 2015), 2 studies did not differentiate between binge and/or purging behaviour (Bleck & DeBate, 2013; Bleck et al., 2015) and another study found that the association was significant only in a subgroup of participants (Muller et al., 2014). Specifically, Muller et al. (2014), investigating temperament subtypes among treatment seeking obese individuals, found that patients who were characterised as “emotionally dysregulated/undercontrolled” showed more ADHD symptoms compared to “resilient/high functioning” patients. The prevalence of BED was also higher in the “emotionally dysregulated/undercontrolled” group compared to the “resilient/high functioning” group (55.9% vs. 27.3%), suggesting a positive association between ADHD and BED in obese “emotionally dysregulated/undercontrolled” adults.

Findings from population studies generally supported a significant positive association between ADHD and BED symptoms (Bleck & DeBate, 2013; Bleck et al., 2015; De Zwaan et al., 2011; Goldschmidt et al., 2015; Hudson et al., 2007; Pagoto et al., 2009; Slane et al., 2010; Sonnevile et al., 2015; Swanson et al., 2011). The 6 studies that did not find a significant association between ADHD and BED and/or BED symptoms recruited children and adolescents who were overweight/obese and hospitalised (Agranat-Meged et al., 2005) or referred for weight reduction (Pauli-Pott et al., 2013) and obese adults (Alfonsson et al., 2012; Davis et al., 2009; Gruss et al., 2012; Muller et al., 2012) who were mainly presenting for bariatric surgery. In general, these studies were limited by very small sample sizes of participants with ADHD (ranging from 8 to 19 individuals). Only one of these 6 studies (Davis et al., 2009) recruited obese adults from a community setting that was not limited by a small sample size.

3.7. ADHD and loss of control over eating (LOC-eating)

Five studies were identified that evaluated an association between ADHD and LOC-eating (see Supplementary material) and significant positive associations were found in all 4 studies that were cross-sectional (Alfonsson et al., 2012; Alfonsson, Parling, & Ghaderi, 2013; Erhart et al., 2012; Reinblatt et al., 2015) and included both sexes.

Pagoto et al. (2010) did not directly measure LOC-eating, but investigated the association between symptoms of ADHD and perceived self-efficacy to control eating in a sample of obese patients who had completed a 16-week clinic-based behavioural weight loss program. In line with the previous findings, Pagoto et al. (2010) reported that individuals who screened positive for adult ADHD reported lower self-efficacy to control their eating compared to controls.

3.8. ADHD and overeating behaviour

Twelve studies reported data relevant to an association between ADHD and overeating (see Supplementary material). Nine studies were cross-sectional (Alfonsson et al., 2012, 2013; Davis, Levitan, Smith, Tweed, & Curtis, 2006; Dempsey et al., 2011; Kim et al., 2014; Pagoto et al., 2010; Patte et al., 2016; Pauli-Pott et al., 2013; Strimas et al., 2008) and 3 used an experimental design (Hartmann, Rief, & Hilbert, 2013; Munsch, Hasenboehler, & Meyer, 2011; Wilhelm et al.,

2011). Two studies included only males, one included only females, and the remaining 9 studies included both sexes. Eating behaviour related to overeating, included eating in the presence of emotional distress (emotional eating), eating in response to the palatability and appearance of food (hedonic eating), external eating, eating as a result of increased susceptibility to feelings of hunger, binge eating and tendency to overeat in the presence of palatable foods, other individuals who are overeating, or other disinhibiting stimuli (disinhibited eating). Significant positive associations between ADHD and overeating behaviours were found in 10 of the 12 studies.

Cross-sectional findings generally pointed to a significant association between ADHD and overeating behaviour. In a large study of > 10,000 children from elementary schools in Korea, Kim et al. (2014), used structural equation modelling to investigate associations between ADHD, dietary behaviour and BMI. ADHD was positively associated with consumption of “unhealthy” food, the number of overeating episodes per week and diet speed. Using a similar approach, Davis et al. (2006), in a sample of adult healthy women from the general population, investigated whether ADHD contributes to the obesity risk profile because it fosters a tendency to overeat. Symptoms of ADHD were positively correlated with aspects of overeating, including emotional eating, external eating and binge eating and overeating correlated with higher BMI. Similarly, in a sample of healthy adult males, a positive association between symptoms of ADHD and overeating behaviour was observed, which positively correlated with BMI (Strimas et al., 2008). Recent findings have confirmed these previous reports and together with binge eating, emotional eating and hedonic eating were found to play a significant role in the association between ADHD symptoms and BMI (Patte et al., 2016).

Eating and/or craving food in response to emotional distress was also found to be positively correlated with ADHD in studies of obese adults (Alfonsson et al., 2012, 2013; Pagoto et al., 2010). However, Pauli-Pott et al. (2013) did not find a significant difference in emotional eating between overweight/obese children and adolescents with clinical and/or sub-clinical symptoms of ADHD and overweight/obese children and adolescents without symptoms of ADHD. The very small number of children/adolescents in the study who met the clinical criteria for ADHD ($n = 17$) together with the questions about emotional eating being limited to feelings of loneliness, disappointment and unhappiness suggests that these findings should be interpreted with caution.

Experimental studies are relatively limited in children and adolescents. Two studies reported significant positive associations between ADHD and laboratory measured food intake (Hartmann et al., 2013; Munsch et al., 2011), whereas another study reported no association (Wilhelm et al., 2011). Methodological differences may explain these inconsistent findings. Thus, Wilhelm et al. (2011), in contrast to the other two research groups (Hartmann et al., 2013; Munsch et al., 2011) assessed food intake of hungry participants after an overnight fast. Therefore, it is possible that overeating tendencies are more reliably investigated after a standardized meal in a satiated state.

3.9. Quality ratings and strength of evidence

Inter-rater agreement for quality assessment was good ($\kappa = 0.78$, 95% CI: 0.64–0.92). Quality ratings varied significantly across studies although most of the studies included were moderate ($n = 44$) quality. Twelve studies were high quality, and the remaining 19 studies were low quality. Small sample sizes, absence of control groups, non-representative sampling procedures and poor or no control of confounds were the main limitations.

Fig. 2 illustrates the strength of evidence available concerning the association between ADHD and disordered eating behaviour. Overall, there is consistent moderate to high strength of evidence that ADHD is associated positively with BN/BN symptoms. There is consistent moderate strength of evidence that ADHD is associated positively with: EDs,

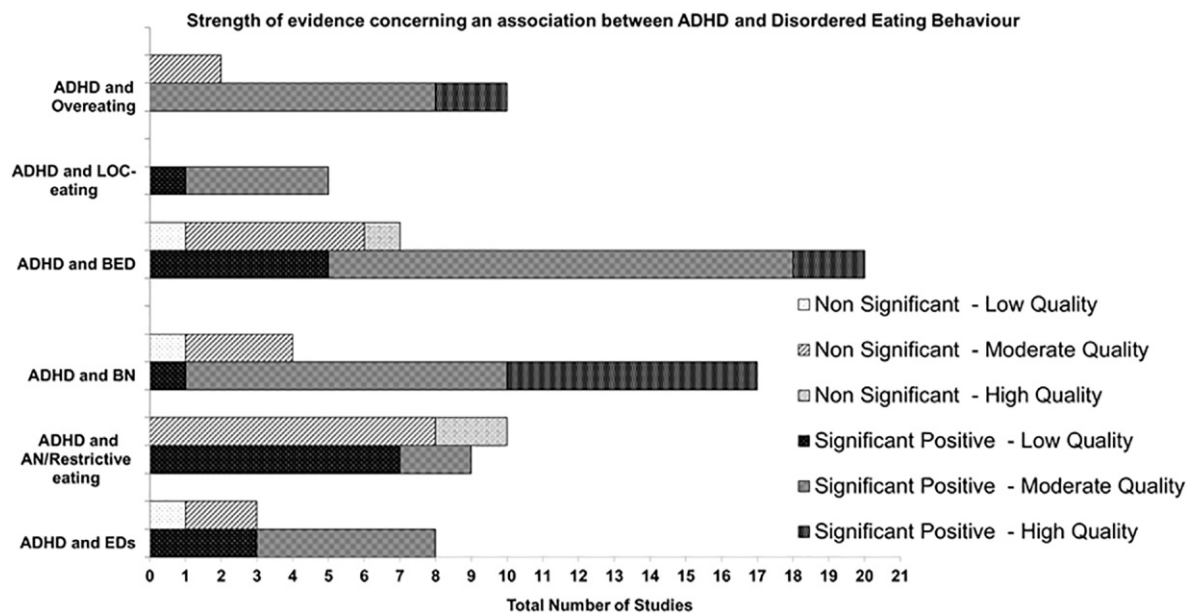


Fig. 2. Strength of evidence concerning an association between ADHD and disordered eating behaviour.

BED/BED symptoms, LOC-eating and overeating behaviour. There is inconsistent evidence whether an association exists between ADHD and AN/Restrictive eating.

3.10. Specific symptoms of ADHD and disordered eating behaviour

Twenty of the 75 studies in this review reported associations between specific symptoms of ADHD and ED types (see Supplementary material). Most studies were cross-sectional studies ($n = 9$) (Alfonsson et al., 2012; Cortese, Isnard, et al., 2007; Ebenegger et al., 2012; Grabarek & Cooper, 2008; Muller et al., 2014; Nazar et al., 2014; Reinblatt et al., 2015; Slane et al., 2010; Yates et al., 2009). Two studies were prospective (Mikami et al., 2010; Mikami et al., 2008), 2 studies were longitudinal (Goldschmidt et al., 2015; Rastam et al., 2013), 2 studies were cohort studies (Khalife et al., 2014; Sonnevile et al., 2015), 1 study used a case-control design (Seitz et al., 2013), 1 study was a case double-control study (Davis et al., 2009), 2 studies were experimental (Munsch et al., 2011; Wilhelm et al., 2011) and 1 was a secondary analysis of data from the US National Longitudinal Study of Adolescents Health (Bleck et al., 2015). Five studies included only females, 1 included only males and the remaining 14 studies included both sexes.

Overall, evidence was mixed, with the exception of studies that assessed the association between impulsivity symptoms of ADHD and BN/BN symptoms which generally found significant positive associations. Mikami et al. (2010, 2008) found that childhood impulsivity symptoms as opposed to hyperactivity and inattention best predicted adolescent BN symptoms at 5 and 8 years follow-up. Cortese, Isnard, et al. (2007) also found indirect evidence that the association between ADHD symptoms and bulimic behaviour in severely obese adolescents can be accounted for mainly by symptoms of impulsivity and inattention but not hyperactivity. However, Seitz et al. (2013) found that inattentive, rather than impulsivity or hyperactivity symptoms of ADHD, explained the severity of BN symptoms, but the recruitment of a clinical population of females seeking treatment for BN limits the generalisability of these findings. The absence of a relationship between hyperactivity symptoms of ADHD and BN/BN symptoms was also a consistent finding among studies that investigated this potential association (Cortese, Isnard, et al., 2007; Mikami et al., 2010; Mikami et al., 2008; Seitz et al., 2013).

Interestingly, hyperactivity symptoms were significantly associated with restrictive eating symptomatology, particularly in men. Grabarek and Cooper (2008) explored sex-based patterns of relationships between symptoms of ADHD and eating pathology and found that hyperactivity (and not inattention) was significantly related to a drive for thinness in men. However, no significant correlation, for either the inattention or the hyperactivity subscale and drive for thinness was found for women. In line with these findings, hyperactivity/impulsivity symptoms were strongly associated with restrictive eating pathology in boys, but not in girls (Rastam et al., 2013).

Findings on the association between inattentive symptoms of ADHD and specific ED subtypes were generally mixed, with the exception of studies that assessed their association with restrictive eating disorders (Yates et al., 2009) and other symptomatology related to restrictive eating (Grabarek & Cooper, 2008; Rastam et al., 2013), which consistently reported non-significant associations. Studies that assessed the relationship between inattentive symptoms of ADHD and BN symptoms or BED symptoms, and reported significant positive associations were cross-sectional, and included clinical populations; specifically eating disorder inpatients (Yates et al., 2009), females seeking treatment for BN (Seitz et al., 2013), obese women seeking non-surgical treatment for obesity or eating disorders (Nazar et al., 2014), bariatric surgery candidates and obese inpatients (Muller et al., 2014) and severely obese adolescents participating in a 6- to 11-month weight loss program (Cortese, Isnard, et al., 2007).

Findings on the association between specific symptoms of ADHD and LOC-eating, although limited, pointed to a significant association between hyperactivity/impulsivity symptoms and LOC-eating (Alfonsson et al., 2012; Reinblatt et al., 2015).

3.11. Moderators and mediators

Twenty-one of 75 studies reported participant characteristics for moderation. Most studies ($n = 18$) investigated the moderating effect of sex on the association between ADHD and disordered eating behaviour (Alfonsson et al., 2013; Bleck & DeBate, 2013; Davis et al., 2015; Grabarek & Cooper, 2008; Mattos et al., 2004; Mikami et al., 2010; Neumark-Sztainer et al., 1995; Patte et al., 2016; Pauli-Pott et al., 2013; Rastam et al., 2013; Slane et al., 2010; Sobanski et al., 2007; Surman et al., 2006; Welch et al., 2015; Yoshimasu et al., 2012). The findings were mixed: eight studies reported a significant effect of sex

(Grabarek & Cooper, 2008; Mikami et al., 2010; Rastam et al., 2013; Sobanski et al., 2007; Surman et al., 2006), while 8 studies found no significant effect (Alfonsson et al., 2013; Davis et al., 2015; Mattos et al., 2004; Patte et al., 2016; Pauli-Pott et al., 2013; Slane et al., 2010; Welch et al., 2015; Yoshimasu et al., 2012). One study found a significant effect for diagnosed EDs but not for disordered eating behaviour (Bleck & DeBate, 2013), and another found a significant sex effect only for use of laxatives, diuretics or emetics for weight loss (Neumark-Sztainer et al., 1995).

When stratified by type of ED, the results were more consistent. Thus, Mikami et al. (2010) found that the positive association observed between childhood ADHD symptoms of impulsivity and parent-reported BN symptoms at 8-year follow-up was stronger for girls than for boys. Surman et al. (2006), in an analysis of two samples of adults, reported significantly greater rates of BN in women with ADHD compared to women without ADHD; however, rates of BN did not differ between men with and without ADHD. Neumark-Sztainer et al. (1995) found that adolescents with ADHD reported more symptoms related to BN pathology than controls. However, only girls with ADHD reported the use of laxatives, diuretics or emetics for weight loss. Findings on the moderating effect of sex on the association between ADHD symptomatology and binge eating generally pointed to a non-significant effect (Davis et al., 2015; Mattos et al., 2004; Patte et al., 2016; Pauli-Pott et al., 2013; Slane et al., 2010). Similarly, Alfonsson et al. (2013) reported no significant gender effect on the relationship between ADHD and LOC-eating, a core feature of binge eating. Five studies assessed the moderating effect of sex on the association between symptoms of ADHD and AN/restraint eating symptomatology (Grabarek & Cooper, 2008; Pauli-Pott et al., 2013; Rastam et al., 2013; Slane et al., 2010; Welch et al., 2015). Findings from adults consistently indicated that hyperactivity symptoms of ADHD were more strongly related to dietary restraint and/or drive for thinness in men than women (Grabarek & Cooper, 2008; Slane et al., 2010). Hyperactivity/impulsivity symptoms of ADHD were also found to be strongly associated with restrictive eating pathology in boys, but not in girls (Rastam et al., 2013). However, in overweight/obese children and adolescents, Pauli-Pott et al. (2013) did not find a significant sex effect on the relationship between ADHD and dietary restraint.

Findings on the moderating effect of age were limited as no studies tested the effect of age using moderation analysis, but 4 studies reported relevant data. Surman et al. (2006) suggest a significant effect of age on the association between ADHD and BN, especially for women. Further, Mikami et al. (2010, 2008) assessed the longitudinal association between ADHD and BN in girls and found that although no girls met full diagnostic criteria for BN at 5 and 8 years follow-up, those with ADHD were more likely to have disordered eating behaviour and BN symptoms.

Three studies reported data on the moderating effect of weight (Erhart et al., 2012; Mikami et al., 2010; Wilhelm et al., 2011) suggesting that the effect of ADHD on eating behaviour does not depend on the weight of the individual.

Other moderators explored included race, punitive parenting, parental expression of emotion (a construct measuring parental criticism of and emotional over-involvement with the child) and peer rejection (Bleck & DeBate, 2013; Mikami et al., 2008). The only study to examine the moderating effect of race, reported that Asian females with ADHD were significantly less likely to have an ED than White females with ADHD (IRR: <0.01; 95% CI: <0.01–<0.01) (Bleck & DeBate, 2013). Punitive parenting, parental expression emotion and peer rejection were assessed as potential moderators by Mikami et al. (2008). Punitive parenting was reported by Mikami et al. (2008) to be a significant predictor of pathological eating in girls with ADHD, but not in controls (significant interaction between ADHD diagnosis and punitive parenting, $B = 0.24$; $p < 0.01$). Interactions between expression of emotion or peer rejection and ADHD were not significant.

Only three of the 75 studies included in this review tested for mediation. Sonnevile et al. (2015), in a sample of children/adolescents

assessed the relationship between ADHD and binge eating, and found that symptoms of ADHD (hyperactivity/inattention) during late childhood were significantly associated with binge-eating in mid-adolescence, and this relationship was mediated via overeating in late-childhood and a strong desire for food in early adolescence. To investigate whether impulsivity can explain the relationship between ADHD symptoms and binge eating, Steadman and Knouse (2014) tested this mediation pathway in undergraduate students. As impulsivity is a multifactorial construct, Steadman and Knouse (2014) used different approaches to measure impulsivity (the Barratt Impulsiveness Scale, the Impulsiveness Questionnaire, the Barkley Deficits Executive, Self-Restraint subscale and a behavioural Go/No-Go task), which are thought to represent different underlying processes. However, none of the measures of impulsivity were found to be significant mediators between ADHD and binge eating symptoms. Davis et al. (2015) using a community-based sample of young men and women found that a high-risk personality profile associated with impulsive and rash responding, and with anxiety proneness may, in part, account for the relationship between ADHD symptomatology and the use/abuse of a broad range of addictive behaviours including food bingeing.

4. Discussion

To the best of our knowledge, this is the first review that has systematically assessed the association between ADHD and disordered eating. Our aim was to evaluate whether there is sufficient evidence to suggest an association between ADHD and disordered eating behaviour, and to examine if ADHD is associated with specific types of disordered eating behaviour. Seventy-five studies were identified and included in this review and overall, the evidence suggests that ADHD is positively associated with disordered eating. However, the strength of evidence is moderate because the majority of studies were limited by methodological issues, including non-representative sampling of participants, small sample sizes, the absence of control groups and poor control for confounds. In addition, there was heterogeneity across studies, particularly in the sample characteristics and the measurement of disordered eating and ADHD, which may confound interpretation of the results. Furthermore, most studies were cross-sectional, and therefore a causal relationship between ADHD and disordered eating cannot be inferred, and the possibility of reverse causality cannot be excluded. The research evidence relating to the effect of ADHD medication on appetite and disordered eating behaviour is beyond the aim of this review. However, medication for ADHD is potentially a significant confounder when assessing a possible association between ADHD and EDs and/or eating pathology/disordered eating. Nevertheless, within the studies reviewed here only five controlled for stimulant medication (Davis et al., 2015; Hinshaw et al., 2012; Pagoto et al., 2009; Reinblatt et al., 2015; Wilhelm et al., 2011), and none of these controlled specifically for the type of stimulant medication, while only one controlled specifically for the dose of stimulant medication (Wilhelm et al., 2011). There is consistent moderate to high strength of evidence that ADHD is positively associated with BN symptoms, and consistent moderate strength of evidence that ADHD is positively associated with eating behaviour related to overeating, including BED symptoms, emotional, external, disinhibited eating and LOC-eating. Evidence for an association between ADHD and AN/Restrictive eating is inconsistent.

Of the studies reviewed only 21 out of the 75 reported participant characteristics for moderation. Sex was one of the commonly reported moderators (Davis et al., 2015; Patte et al., 2016; Pauli-Pott et al., 2013; Slane et al., 2010). Although all of the studies that stratified by sex provide evidence for a stronger association between ADHD and BN/BN symptoms in females than males, it is unclear whether the associations found between sex and BN/BN symptoms are unique to ADHD or simply indicate general population patterns of differential sex prevalence of BN/BN symptoms. In addition, although it is consistently reported that the association between hyperactivity symptoms of ADHD and

restrained eating symptomatology is stronger for males than females, it is plausible that this is a reflection of the higher hyperactivity symptoms in males compared to females (Gaub & Carlson, 1997; Williamson & Johnston, 2015) and not a higher predisposition for males with ADHD to be diagnosed with restrained eating. There is currently insufficient evidence to determine whether age, weight and ADHD medication moderate the relationship between ADHD and disordered eating behaviour.

Mechanism testing was very limited in the studies reviewed and only three of the 75 studies tested for mediation (Davis et al., 2015; Sonnevile et al., 2015; Steadman & Knouse, 2014). Sonnevile et al. (2015) found that the association between ADHD and binge eating in mid-adolescence was mediated by childhood over-eating and strong desire for food in early-adolescence, suggesting that early childhood ADHD symptoms in, in addition to an overeating phenotype, may contribute to risk for adolescent binge eating via a strong desire for food in early adolescence. However, several other pathways could explain the association between ADHD and disordered eating behaviour.

It is conceivable that disordered eating behaviours are not directly related to ADHD and may be mediated by other, often common, co-existing health-related conditions, such as depression and other mood disorders (Austerman, 2015) and EDs (Blinder et al., 2006; Casper, 1998; Keel, Klump, Miller, McGue, & Iacono, 2005). Adjustment for comorbid conditions that are common in ADHD was limited in the studies reviewed and only 8 of the 75 studies reported an adjusted measure of association. Of these studies a significant association between pathological eating and ADHD, persisted after adjusting for a variety of covariates, including depression, anxiety, mood, antisocial behaviour, stage of development and internalizing and externalizing conditions (Biederman et al., 2010; Cortese, Isnard, et al., 2007; Mikami et al., 2008; Reinblatt et al., 2015), suggesting that ADHD is a specific risk factor for eating pathology.

Alternatively, biological factors such as shared common genetic variants could partly explain the link between ADHD and disordered eating. For example, dysfunctions in the dopamine pathways of the brain have been found among both individuals who are obese (Val-Laillet et al., 2015) and individuals with ADHD (Badgaiyan, Sinha, Sajjad, & Wack, 2015). Recent findings also suggest that individuals who have ADHD symptoms and carry genetic profiles associated with greater dopamine activation in brain reward areas are more likely to engage in overeating behaviour, such as binge, emotional and hedonic eating (Patte et al., 2016).

Personality factors should also be considered. An avoidant coping style has been associated with impaired eating behaviour (Martyn-Nemeth, Penckofer, Gulanick, Velsor-Friedrich, & Bryant, 2009; Troop, Holbrey, Trowler, & Treasure, 1994). Furthermore, eating psychopathology has been associated with decreased use of adaptive coping strategies (e.g., problem-focused coping) (Mayhew & Edelmann, 1989; Troop et al., 1994). Therefore, the association between ADHD and disordered eating may be complex and compounded by feedback loops including an ability to cope with challenges in life.

It is also plausible that core processes underlying ADHD give rise to pathological eating patterns. Impulsivity, a main feature of ADHD has been found to be a characteristic of patients with BN and BED (Dawe & Loxton, 2004; Fischer, Smith, & Anderson, 2003; Penas-Lledo & Waller, 2001). Four of the studies reviewed reported associations between impulsivity symptoms of ADHD and BN/BN symptoms (Cortese, Isnard, et al., 2007; Mikami et al., 2010; Mikami et al., 2008; Seitz et al., 2013) and there is evidence that impulsivity symptoms of ADHD are positively associated with BN symptoms and with BED (Muller et al., 2014). Hyperactivity/impulsivity symptoms of ADHD were also consistently positively associated with LOC-eating, a core symptom of BED (Alfonsson et al., 2012; Reinblatt et al., 2015). Impulsivity has been suggested to positively influence overeating (Jasinska et al., 2012; Meule, 2013; Nederkoorn, Braet, et al., 2006; Nederkoorn, Smulders, et al., 2006), as it may increase the susceptibility that highly palatable food-

cues attract attention and thereby trigger eating behaviour (Castellanos et al., 2009; Hou et al., 2011; Polivy, Herman, & Coelho, 2008). Therefore, impulsivity symptoms of ADHD may relate to binge eating, and the binge eating episodes of BN, via an enhanced susceptibility to food-cues.

Inattention symptoms of ADHD may also be associated with disordered eating and it has been suggested that compulsive eating may be a compensatory mechanism to help control frustration associated with attention and organization difficulties (Schweickert, Strober, & Moskowitz, 1997). Davis et al. (2006) proposed that patients with ADHD may be inattentive to internal signs of hunger and satiety. Thus, they may forget about eating when they are engaged in other activities and may be more likely to eat when less stimulated, at which point they may be very hungry. Eating in response to feelings of hunger and satiety has been found to be associated with BMI (Gast, Campbell Nielson, Hunt, & Leiker, 2015; Madden, Leong, Gray, & Horwath, 2012), suggesting that inattention to internal signs of hunger and satiety may cause overeating. However, the evidence to date concerning the association between inattention symptoms of ADHD and disordered eating is mixed. In this review, the studies that found significant positive associations were cross-sectional in nature. Therefore, reverse causality may also be possible, as nutritional deficiencies that are often common in EDs (Mitchell & Crow, 2006; Setnick, 2010) may impact on a patients' ability to focus attention, resulting in symptoms that mimic attention deficits in ADHD.

The role of hyperactivity symptoms of ADHD in disordered eating has generally been neglected. However, in this review, hyperactivity symptoms of ADHD were found to be significantly associated with restrictive eating behaviours, particularly in men (Grabarek & Cooper, 2008; Rastam et al., 2013; Slane et al., 2010). Excessive exercise is often characteristic of patients suffering from AN, especially in the acute phase of the disorder (Kohl, Foulon, & Guelfi, 2004). Therefore, the observed association between hyperactivity symptoms of ADHD and restrictive eating symptomatology may be mediated by (over)exercising behaviour.

4.1. Strengths and limitations of the systematic review

This systematic review has a number of strengths and some limitations. Eating behaviour was conceptualised as a continuum ranging from normal eating to eating disorders, and therefore a range of eating behaviour was included. ADHD was also conceptualised dimensionally, allowing a large number of studies to be systematically reviewed. Certain limitations require that the results of this review should be interpreted with some caution. Most studies published in this area were not specifically designed to explore an association between ADHD and disordered eating. In many cases this has resulted in suboptimal study designs, with the potential for biased results. Language and publication bias may also be relevant, as the search was limited to studies published in the English language. Finally, due to the heterogeneity of the studies, particularly with respect to methodologies, outcomes and populations, a meta-analysis was not feasible.

4.2. Clinical implications

EDs can significantly impact both the physical and the psychological health of individuals, and thus it may be advisable to assess the risk for disordered eating behaviour in the management of ADHD. Recent research has focussed on the association between ADHD and obesity (Cortese et al., 2015), but the current findings highlight the importance of considering the risk that individuals with ADHD may develop a range of disordered eating behaviour. Individuals with ADHD can present with normal weight, but suffer from eating pathologies (Fairburn & Cooper, 1982). Furthermore, hyperactivity symptoms of ADHD in children may be protective against obesity, leading to an underestimation of problematic eating behaviours that could co-exist with ADHD. However, as symptoms of ADHD manifest differently with age, and symptoms of hyperactivity typically decrease and become more subtle (Holbrook

et al., 2016; Todd et al., 2008) the continuation of problematic eating behaviour can result in the development of overweight/obesity later in adolescent/adult life due to an age-related decrease in energy expenditure. Therefore, weight management issues need to be taken into account when considering medication for ADHD. Recently, lisdexamfetamine, a central nervous system stimulant used for the treatment of ADHD, has been approved for the treatment of moderate to severe BED (US Food and Drug Administration, 2015). It is unclear if this medication would be effective for individuals with ADHD who exhibit restrictive eating, and assessment of eating behaviours would be an important factor to consider when making treatment decisions. Ultimately, a better understanding of the range of specific eating problems experienced by individuals with ADHD and their underlying mechanisms will facilitate more effective and personalised treatment.

4.3. Future research

Research on the association between ADHD and disordered eating is in its infancy and some methodological limitations of previous studies could be addressed in future research. In this review, assessment of eating pathologies varied substantially across studies, even among studies of individual EDs, making comparisons across samples difficult. Studies used DSM criteria, symptom counts, ED questionnaires, or single questions about eating behaviours, while few studies assessed eating behaviour in a laboratory setting. Use of uniform and robust methods of assessing disordered eating would be preferable in future studies. As most studies in this review included a population comprised mainly or only of females, future studies would benefit from adequate representation of both sexes in samples. This would enable a better understanding of the role of sex in the association between ADHD and EDs and/or eating pathology/disordered eating.

Confounding factors such as health characteristics, stimulant medication and ADHD-related comorbidities should be included in the models in addition to the confounds already identified in previous research (e.g., age, sex, socioeconomic status). Drug therapies for ADHD such as methylphenidate can have a pronounced effect on appetite (Findling et al., 2008), while lisdexamfetamine is clinically effective in treating both ADHD and BED (Brownley et al., 2016). To disentangle the effect of ADHD from any effect of stimulant medication on disordered eating, future studies should control for factors such as type of stimulant medication, length of stimulant treatment and medication dosage.

Further research is also required to clarify the nature of the mechanisms underlying the association between ADHD and disordered eating. An emerging question is whether the relationship between ADHD and disordered eating is influenced by conditions that often co-exist with EDs and eating pathologies, including anxiety and depression or whether core features of ADHD may lead to pathological eating behaviour. Given the extensive body of evidence that suggests an association between impulsivity symptoms of ADHD and BN symptoms, future studies should address the role of core processes such as impulsivity in disordered eating. As impulsivity involves at least two identifiable cognitive/emotional processes, reward-driven behaviours and poor inhibition (Solanto et al., 2001), future research should address whether food-cue induced overeating may be the result of overwhelming, reward-related, bottom-up processes or deficient inhibitory, top-down control mechanisms, or both (Appelhans, 2009; Heatherton & Wagner, 2011; Price, Higgs, Maw, & Lee, 2016). Further studies are also needed to explore whether inattention and hyperactivity symptoms may contribute to disordered eating and the underlying processes of any relationship.

We propose a research framework to guide future studies on ADHD and disordered eating based on the National Institute of Mental Health Research Domain Criteria Initiative (RDoC), which encourages research on dimensions of observable behaviour and neurobiology rather than a categorical, symptom-based approach to the study of mental health (Insel et al., 2010). Our proposed research framework comprises multi-modal, laboratory-based assessment of cognitive constructs

across different RDoC systems. Studying the relationship between individual variations in specific RDoC cognitive constructs of attention and cognitive control and disordered eating in ADHD provides an opportunity to identify core processes that cut across diagnostic categories and could be targeted by therapeutic interventions. We propose recruitment of participants from the community to span the range of variation in cognitive processes associated with ADHD. This dimensional approach ensures that potential confounds associated with clinical research (e.g., medication status) can be minimised. Participants would complete behavioral tasks that assess specific cognitive constructs including cognitive control and attention as well as questionnaire items assessing ADHD symptoms. In addition, functional brain imaging could provide a link from behavioral and self-report measures to neural units of analysis. Disordered eating behaviour would be assessed by both self-report measures and laboratory based assessment of loss of control over eating. Tanofsky-Kraff et al. (2013) have proposed a similar approach to the study of disinhibited eating in obese adolescents but an RDoC compatible approach has yet to be adopted widely in the study of disordered eating. While high quality longitudinal studies are the gold standard for understanding the temporal relationship between ADHD and disordered eating behaviour, such designs are often impractical and unfundable. Our proposed framework enables testing for causal relationships between cognitive constructs and disordered eating because processes such as attention and cognitive control can be manipulated and the effects on laboratory measures of eating (such as measures of eating rate captured using a Universal Eating Monitor) assessed. This framework could also be used to study disordered eating in other mental health conditions associated with disordered eating such as schizophrenia and depression and may be extended from cognitive constructs to other RDoC domains and their interaction. For example, it is likely that cognitive control systems and positive valance systems interact to influence food responsiveness (Higgs, 2016; Wildes & Marcus, 2015).

5. Conclusions

There is a consistent moderate strength of evidence that ADHD is positively associated with disordered eating and with specific types of disordered-eating behaviour, in particular, overeating behaviour. In addition, there is evidence that impulsivity is positively associated with BN symptoms. There is also more limited evidence to suggest an association between hyperactivity symptoms and restrictive eating in males but not females, although this requires further investigation. Increased awareness of this phenomenon could enhance clinical management and therapeutic options for individuals with ADHD. Greater understanding of the relationship between core cognitive constructs and disordered eating behaviour potentially using the RDoC framework will be useful for informing therapeutic options for individuals with ADHD and other mental health conditions that are associated with eating disorders.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at <http://dx.doi.org/10.1016/j.cpr.2017.03.002>.

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Chapter 4:
Associations Between Core Symptoms of Attention Deficit Hyperactivity Disorder and
Both Binge and Restrictive Eating

4.1 Abstract

It is unclear whether core symptoms of attention-deficit/hyperactivity disorder (ADHD) relate to specific types of disordered eating behaviour. In addition, very few studies to date have investigated potential mediators linking ADHD and disordered eating. We investigated associations between core symptoms of ADHD and disordered eating, including both binge/disinhibited eating and restrictive eating behaviour, and assessed whether negative mood and/or deficits in awareness and reliance on internal hunger/satiety cues mediate these relationships. In two independent studies, we used a dimensional approach to study ADHD and disordered eating. In Study 1, a community-based sample of 237 adults (72.6% female, 18-60 years [$M = 26.8$, $SE = 0.6$]) completed an online questionnaire, assessing eating attitudes and behaviours, negative mood, awareness and reliance on internal hunger/satiety cues and ADHD symptomatology. In Study 2, 142 students (80.3% female, 18-32 years [$M = 19.3$, $SE = 0.1$]) were recruited to complete the same questionnaires in the laboratory, and complete tasks assessing interoceptive sensitivity and impulsivity. In each study, core symptoms of ADHD correlated positively with both binge/disinhibited and restrictive eating and negative mood mediated the relationships. Deficits in awareness and reliance on internal hunger/satiety signs also mediated the association between inattentive symptoms of ADHD and disordered eating, especially binge/disinhibited eating. We provide the first evidence for a direct relationship between inattentive symptoms of ADHD and binge/disinhibited eating. Further investigation of the role of inattentive symptoms of ADHD in disordered eating may be helpful in developing novel treatments for both ADHD and binge eating.

4.2 Introduction

Attention Deficit Hyperactivity Disorder (ADHD) is defined as a persistent pattern of inattention and/or hyperactivity/impulsivity that is more frequently displayed and is more severe than is typically observed in individuals at a comparable level of development (American Psychiatric Association, 2013). Prevalence rates of ADHD in childhood are estimated to be as high as 15.5% (Rowland et al., 2015), and in approximately 75% of cases, symptoms of ADHD do not remit in childhood/adolescence and continue into adulthood (Biederman, Petty, Clarke, Lomedico, & Faraone, 2011). Existing research links ADHD with a number of adverse outcomes (Instanes, Klungsoyr, Halmoy, Fasmer, & Haavik, 2016; Wilens et al., 2007), and accumulating evidence suggests that there is a strong association between ADHD and eating disorders (EDs)/disordered eating (Kaisari, Dourish, & Higgs, 2017; Levin & Rawana, 2016; Nazar et al., 2016). Findings from recent studies suggest that specific symptoms of ADHD may relate to disordered eating (Cortese et al., 2007; Davis, Levitan, Smith, Tweed, & Curtis, 2006; Strimas et al., 2008; Tong, Shi, & Li, 2017; Yilmaz et al., 2017), and individuals with high symptoms -without necessarily meeting criteria for a clinical diagnosis- have been found to be at increased risk of disordered eating compared to individuals with low symptoms of ADHD (Yilmaz et al., 2017). Despite this evidence base, it remains unclear whether core symptoms of ADHD relate to specific subtypes of disordered eating.

Disordered eating refers to patterns of eating behaviour that deviate from normal but do not meet all the criteria for a clinical diagnosis of an ED. Although the recorded ED prevalence rate is around 0.5–3%, depending on the specific ED diagnosis, disordered eating symptomatology in the general population has been found to be as high as 12% (Nagl et al., 2016). This is a concern not only because of the associated psychopathology, but also because

disordered eating is a risk factor for full-syndrome eating disorders (Goldschmidt, Aspen, Sinton, Tanofsky-Kraff, & Wilfley, 2008). It is therefore important to explore risk factors for disordered eating, such as ADHD symptomatology, in the general population. Similarly, the National Institute of Mental Health Research Domain Criteria Initiative (RDoC) encourages research on dimensions of observable behaviour rather than a categorical, symptom-based approach to the study of mental health (Insel et al., 2010).

Impulsivity symptoms of ADHD have been consistently positively associated with bulimic and binge eating behaviour (Cortese et al., 2007; Mikami et al., 2010; Mikami, Hinshaw, Patterson, & Lee, 2008; Muller, Claes, Wilderjans, & de Zwaan, 2014). For instance, Mikami et al. (2010, 2008) found that childhood impulsivity symptoms as opposed to hyperactivity and inattention best predicted adolescent bulimia nervosa symptoms at 5 and 8 years follow-up. In addition, hyperactivity/impulsivity symptoms of ADHD were found to be positively associated with loss of control over eating, a core symptom of binge eating disorder (Alfonsson, Parling, & Ghaderi, 2012; Reinblatt et al., 2015). However, the evidence to date for an association between inattentive symptoms of ADHD and EDs/disordered eating is mixed and further research is required to assess the specific contribution (*if any*) of inattentive symptoms of ADHD in disordered eating (for a review see Kaisari et al., 2007).

It is plausible that mood changes associated with depression, anxiety and stress mediate the relationship between ADHD and risk for disordered eating. Indeed, several studies have reported that depression and anxiety are the most frequently reported psychiatric comorbidities in ADHD patients (Friedrichs, Igl, Larsson, & Larsson, 2012; Hodgkins, Montejano, Sasane, & Huse, 2011; Kessler et al., 2006). A strong positive association between ADHD symptoms in adulthood and levels of self-perceived stress has also been reported (Hirvikoski, Lindholm, Nordenstrom, Nordstrom, & Lajic, 2009; Lackschewitz,

Huth, & Kroner-Herwig, 2008). Such data suggest that eating pathology may be a form of “self-medication”, and a means to cope with frustrations and negative affect caused by the outcomes of attention difficulties (e.g. poor work performance) and/or impulsive responses at the cost of planned goals. Life challenges experienced by individuals with symptoms of ADHD may also lead to generalised feelings of perceived lack of control. Issues of control have been proposed to play a central role in the aetiology and maintenance of EDs, in particular anorexia nervosa (AN) (Bruch, 2001; Fairburn, Shafran, & Cooper, 1999). Thus, it has been suggested that through exerting control over eating, patients with AN, attempt to avoid negative affect associated with ineffectiveness and lack of control experienced in the rest of the individual’s life (Slade, 1982). Taken together, this evidence indicates that symptoms of ADHD may relate to both binge/disinhibited eating and restrictive eating pathology indirectly via negative mood. To the best of our knowledge no studies to date have investigated the potential mediating role of negative mood in the hypothesised associations between ADHD symptomatology and eating pathology.

Another possibility is that the relationship between ADHD and disordered eating is mediated by lack of awareness of internal signs of hunger and satiety, which could contribute to a disturbed pattern of eating behaviour. For instance, individuals with high inattentive symptoms of ADHD may forget about eating when engaged in other activities, leading to subsequent overeating or lack of control over eating (Fleming & Levy, 2002). A lack of awareness and knowledge of internal signs of hunger and satiety may also contribute to restrictive eating pathology if external control such as restricting food intake is used to override internal signals to adhere to a culturally imposed thin-ideal stereotype (Augustus-Horvath & Tylka, 2011; Avalos & Tylka, 2006). Hence, core symptoms of ADHD may contribute to both binge/disinhibited eating and restrictive eating indirectly by a lack of

awareness of internal signs of hunger and satiety. Indeed, the link between eating in response to internal signs of hunger and satiety and disordered eating is also evident from studies of individuals with eating disorders (Fassino, Piero, Gramaglia, & Abbate-Daga, 2004; Klabunde, Acheson, Boutelle, Matthews, & Kaye, 2013; Pollatos et al., 2008; van Dyck, Herbert, Happ, Kleveman, & Vogele, 2016). Despite this evidence, to the best of our knowledge no mediational studies have been conducted to date to assess whether awareness and reliance on internal hunger/satiety signs provides a pathway of association between ADHD symptomatology and disordered eating.

Current Research

The current research investigated the potential associations between core symptoms of ADHD and disordered eating, including both binge/disinhibited eating and restrictive eating behaviour, and examined whether negative mood and/or deficits in awareness and reliance on internal hunger/satiety cues might mediate these relationships. Adult men and women, differing in severity of ADHD symptoms were eligible for participation. This approach is predicated on the assumption that personality factors and symptoms of a disorder are best conceptualised dimensionally and occur with normal variation in the general population (Claridge & Davis, 2013). Furthermore, this dimensional approach ensures that potential confounds associated with clinical research (e.g. medication status) can be minimised.

Two studies were conducted: Study 1 was an online study, which investigated the hypothesised mediational pathways of association between hyperactivity/impulsivity and inattentive symptoms of ADHD and disordered eating via negative mood and/or deficits in awareness and reliance on internal hunger/satiety cues. Study 2 was a laboratory study which investigated whether the findings from Study 1 could be replicated in an independent sample,

and assessed the specific contributions (*if any*) of hyperactivity and impulsivity to disordered eating.

4.3 Study 1

The primary aims of Study 1 were: (i) to investigate the associations between core symptoms of ADHD and disordered eating, including both binge/disinhibited eating and restrictive eating behaviour, and (ii) to test whether negative mood and/or deficits in awareness and reliance on internal hunger/satiety cues might mediate these relationships, controlling for important potential confounders such as gender, BMI and current ADHD medication use, that previous research has suggested to relate both to disordered eating and/or ADHD. The moderating effect of age, gender, BMI and ADHD medication in any relationship between core symptoms of ADHD and disordered eating was also investigated. We recruited participants from the general population to take part in an online study.

4.3.1 Materials and Methods

Participants and Procedure

Participants were recruited from the general population, through social media (e.g. Facebook), postings on web sites such as ADHD support groups, and advertisements at the University of Birmingham, UK. Adult men and women, aged 18-60 years, differing in severity of ADHD symptoms were eligible for this study. Individuals receiving medication for ADHD treatment were not excluded but medication status was controlled for in the analysis. Participants were required to be fluent in English. From November 2015 to March 2016, a total of 265 individuals completed an online survey.

All participants provided online informed consent before participation. After informed consent was obtained, participants were directed to a URL to complete the online survey. Participants completed self-report questionnaires and provided demographic information. The

survey took approximately 45 minutes to complete. All participants were informed that they could enter a prize draw to win a £50 gift voucher for their participation. The protocol was approved by the University of Birmingham Research Ethics Committee.

Measures

Data quality. Due to the self-administered nature of online surveys, measurement error is a common problem. In particular, “satisficing”—which involves a respondent exerting minimal cognitive effort in order to quickly complete the survey—erodes the quality of survey data, which may lead to biased results. In the present study, certain techniques were used to allow a quality assessment of the collected responses. These are described in brief below:

Trap questions. Trap questions (e.g. For data quality purposes, please select 4) were used every 50 to 100 survey items (3 in total), as suggested by Meade and Craig (2012), to identify participants who were not paying sufficient attention to instruction and not providing consistent responses.

Self-report of study engagement. At the end of survey, participants were asked to self-report the quality of the data they provided (Meade and Craig, 2012). Specifically, they were asked to respond to the following question: “Finally, it is vital to our study that we only include responses from people who devoted their full attention to this study. In your honest opinion, should we use your data?”. It was emphasised that participants could still enter the prize draw to win the gift voucher for their participation regardless of how they responded to the question.

To minimise measurement error, which may lead to biased results it was decided a priori that responses from participants who answered all three of the trap questions incorrectly

would be removed from any further analysis. A similar approach was followed for participants who responded negatively to the self-report measure of study engagement.

Demographics

Demographics including age, sex, ethnicity, education level and professional status were recorded. Body mass index (BMI; kg/m^2) was calculated based on self-reported measures of height and body weight. A proxy measure of socio-economic status (SES) was calculated based on the participants' responses about their highest education level completed and their current profession. The education level was collapsed into six categories, including some secondary or high school education (including O Levels and GCSEs); A levels, high school diploma, high school certificate, etc.; vocational qualifications; University or college graduate; Master's degree, post-graduate certificate or diploma, and professional or doctoral degree. Profession status was categorised as: unemployed; student; semi-skilled or unskilled manual worker; skilled manual worker; supervisor or clerical, junior managerial, administrative or professional; intermediate managerial, administrative or professional; higher managerial, administrative or professional. A 6- and 7-point scale was used to score the education and profession variables respectively, with higher scores representing higher education level completed and higher managerial, administrative or professional status. The SES was calculated as the mean score of the two variables.

Current Symptoms of ADHD

The Conners' Adult ADHD Rating Scale-Self Report Screening Version (CAARS-S: SV) (Conners, Erhardt, & Sparrow, 1999), was used to assess current ADHD symptoms. The CAARS is a suitable instrument for evaluating ADHD symptoms in adults (Ghassemi, Moradi, Tehrani-Doost, & Abootalebi, 2010) and utilises a 4-point format in which respondents are asked to rate items pertaining to their problems. The self-report screening

version (CAARS-S: SV) which was used in the present study, has 30 items that assess ADHD symptoms according to the 4th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV). The subscales derived from the questionnaire are: (a) Inattentive Symptoms (9 items); (b) Hyperactive-Impulsive Symptoms (9 items); (c) Total ADHD Symptoms; (d) ADHD Index (12 items).

Participants were also asked to report whether they had been diagnosed with ADHD by a healthcare professional. If they answered in the affirmative, they were asked at what age the diagnosis took place, and if they were currently taking medication as part of their treatment protocol or if they received medication during the previous year. If medication was taken, the participant was asked to report the type of medication (e.g. stimulant). ADHD medication use was coded as a dichotomous variable (currently taking medication for ADHD; currently taking no medication for ADHD). Other psychological/psychiatric comorbidities were categorised as present, including any other psychological/psychiatric disorders diagnosed by a healthcare professional apart from ADHD and/or anxiety, depression and eating disorders, or not present.

Disordered Eating

Several established measures of disordered eating were included in the survey:

The Dutch Eating Behaviour Questionnaire (DEBQ) (Van Strien, Frijters, Bergers, & Defares, 1986), a 33-item self-report questionnaire, was used to assess three aspects of eating behaviour: “emotional eating” (*i.e. the degree to which eating is prompted by emotional states like tension and worry rather than by hunger*) (Factor I - 13 items), “external eating” (*i.e. the degree to which one tends to overeat if food looks and smells good*) (Factor II - 10 items) and “dietary restraint” (*i.e. the degree to which one tends to restrict food intake in order to control body weight*; (Factor III – 10 items). These subscales have been shown to

have good reliability and validity. Cronbach's alpha in a sample of 653 women and 517 men ranged from 0.80 to 0.95 across scales and groups (Van Strien et al., 1986). In the present study, the Cronbach's alpha was 0.96, 0.89 and 0.93 for the "emotional eating", "external eating" and "dietary restraint" subscales, respectively.

The Loss of Control over Eating Scale (LOCES) (Latner, Mond, Kelly, Haynes, & Hay, 2014), short version, a 7-item scale, was used to assess subjective perceptions of being compelled to eat or unable to resist or stop eating, resulting in initiating eating when not intended, and/or eating more than originally intended, and/or difficulty stopping eating. The 7-item LOCES utilizes a 5-point scale and responders are asked to rate how often in the last 4 weeks (28 days) they experienced each one of the 7 specified items during a time when they were eating (*e.g. my eating felt like a ball rolling down a hill that just kept going and going*). The tool has been shown to have good test-retest reliability ($r = 0.82$, $p < 0.001$) and validity (Cronbach's $\alpha = 0.93$). In the present study, the Cronbach's alpha was 0.95.

The Binge Eating Scale (BES) (Gormally, Black, Daston, & Rardin, 1982), a 16-item self-report questionnaire, was used to assess binge eating pathology. The BES does not specify a time frame and presents a series of differently weighted statements for each item, from which respondents select the statement that best describes their attitudes and behaviours. This yields a continuous measure of binge eating pathology of 0–46. The BES has good test-retest reliability ($r = 0.87$, $p < 0.001$) and moderate associations with binge eating severity as measured by food records ($r = 0.20$ – 0.40 , $p < 0.05$; (Timmerman, 1999). The Cronbach's alpha in the present study was 0.92.

The Bulimic Investigatory Test, Edinburgh (BITE) (Henderson & Freeman, 1987), is a 33-item self-report questionnaire, designed as an objective screening test to identify subjects with bulimic symptoms. The BITE consists of two subscales: the symptoms scale (30 items),

which measures the level of the symptoms, and the severity scale (3 items), which provides a measure of the frequency of the symptoms. Results from the symptoms scale were analysed in this study. The symptoms scale score ranges from 0-30. Internal consistency of the BITE as assessed by Cronbach's alpha for the symptom scale is high (0.96) (Henderson & Freeman, 1987). In the present study, the Cronbach's alpha was 0.92. The scale has been validated in women with eating disorders, and male and female adults and adolescents (Henderson & Freeman, 1987; Ricciardelli, Williams, & Kiernan, 1999).

The Eating Attitudes Test (EAT-26) (Garner, Olmsted, Bohr, & Garfinkel, 1982), a 26-item self-report questionnaire, is a screening instrument widely used to measure symptoms and characteristics of eating disorders. Furthermore, it has been widely applied as an index of the symptoms of Anorexia Nervosa. The EAT-26 comprises three subscales, Dieting (Factor I - 13 items), Bulimia and Food preoccupation (Factor II - 6 items), and Oral Control (Factor III - 7 items), which are summed to obtain a total score. Whereas bulimic and restricter anorexia nervosa patients were not found to differ on the total EAT-26, these groups did differ on Factors II and III of the EAT-26 (Garner et al., 1982). The tool has been used in both clinical and non-clinical settings and has been found to have strong psychometric properties (Anstine & Grinenko, 2000; Garner et al., 1982; Mintz & O'Halloran, 2000). The Cronbach's alpha measures in the present study were 0.88, 0.87, 0.82 and 0.66 for the EAT Total score, and the Dieting, Bulimia, and Oral Control subscales, respectively.

Awareness and Reliance on Internal Hunger/Satiety Cues

The Reliance on Internal Hunger/Satiety Cues subscale (RIH) of the 21-item Intuitive Eating Scale (IES) (Tylka, 2006), was used to assess awareness and use of internal hunger/satiety cues to guide one's eating behaviour, i.e. to determine when and how much to eat. The RIH comprises 6 items (e.g. "I can tell when I'm slightly hungry"), and item

responses are rated on a scale that ranges from 1 (strongly disagree) to 5 (strongly agree). After appropriate items are reverse-scored, item responses are averaged to arrive at a total score. Higher total scores correspond with higher levels of awareness and use of internal hunger/satiety cues to determine one's eating behaviour. The RIH subscale has been found to be positively correlated with interoceptive sensitivity, as assessed by the heartbeat perception task (Herbert, Blechert, Hautzinger, Matthias, & Herbert, 2013). In the present study, the Cronbach's alpha was 0.85.

Negative Mood

Negative mood was modelled as a latent variable comprising three emotional factors associated with anxiety and depression, and with stress perception: Anxiety and Depression levels, were assessed by the 14-item Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983). The HADS utilises a 4-point Likert scale (range 0–3), and includes 7 items for each subscale. The total score is the sum of the 14 items, and for each subscale the score is the sum of the respective seven items (ranging from 0–21). In a review of the literature, Bjelland et al. (2002) found that the HADS is a valid tool to measure anxiety and depression levels in the general population and performs well in screening for the separate dimensions of anxiety and depression. The Cronbach's alpha in the present study was 0.85 and 0.82 for the Anxiety, Depression subscales, respectively. To assess perception of stress participants were asked to complete the 10-item Perceived Stress Scale (PSS; Cohen, Kamarck, & Mermelstein, 1983). The PSS utilizes a 4-point format in which respondents are asked to report their feelings and thoughts during the last month. The PSS has been validated in numerous studies, including cross-cultural validation, and has consistently shown high internal validity (Cronbach's alpha > 0.70) and good test–retest reliability (Rho > 0.70)

(Leung, Lam, & Chan, 2010; Mimura & Griffiths, 2008). In the present study, the Cronbach's alpha was 0.90.

Alcohol and Drug Use

Alcohol abuse was assessed by the Short Michigan Alcohol Screening Test (SMAST) (Selzer, Vinokur, & van Rooijen, 1975). The SMAST is a 13-item self-report scale where participants are instructed to answer number of questions concerning their alcohol use during the past 12 months with "YES" or "NO". Each "YES" answer equals one point. A score of 3 indicates a borderline alcohol problem and a score of 4 or more indicates that there may be an alcohol problem. Evaluation data indicate that it is an effective diagnostic instrument, and does not have a tendency for false positives. In the present study, the Cronbach's alpha was 0.73. Drug abuse was assessed by the Drug Abuse Screening Test-10 (DAST-10) (Skinner, 1982), a 10-item self-report scale that consists of items that parallel those of the Michigan Alcoholism Screening Test (MAST). The DAST-10 is a short version of the original DAST-28 and has exhibited valid psychometric properties for use as a clinical or research tool in a variety of populations (Yudko, Lozhkina, & Fouts, 2007). In the present study, the Cronbach's alpha was 0.68. Twelve participants had missing data; therefore, the available sample size for the internal consistency reliability analysis was limited to 225 participants.

Analysis Plan

Data Reduction. To condense the information contained in the disordered eating variables into a smaller set of new composite dimensions, with a minimum loss of information, a principal component analysis with varimax rotation was applied, using (SPSS). Principal component analysis revealed that measures of disordered eating loaded onto two components (see Table 1 for component loadings). Together the components explained 73% of the variance observed. The first component (eigenvalue = 5.03), which accounted for 56%

of the variance, was labelled ‘Binge/Disinhibited Eating’. The second component (eigenvalue = 1.56) accounted for a further 17% of the variance and was labelled ‘Restrictive Eating’. A high score on the ‘Binge/Disinhibited Eating’ component is related to a pattern of behaviour that involves a tendency towards overeating and eating opportunistically. This can include eating in response to negative affect, overeating in response to the palatability of food, and not being able to resist temptation to eat despite negative consequences. A high score on the ‘Restrictive Eating’ component is related to a pattern of behaviour to limit food intake as a means of controlling body weight.

Table 1

Component Loadings for Principal Component Analysis of Disordered Eating Variables

	Component 1: Binge/Disinhibited Eating	Component 2: Restrictive Eating
BITE	0.929	
BES	0.926	
LOCES	0.897	
DEBQ-Emotional Eating	0.830	
DEBQ-External Eating	0.739	
EAT-Bulimia & Food Preoccupation	0.725	
EAT-Oral Control		0.728
EAT-Dieting		0.703
DEBQ-Restraint Eating		0.532

Negative mood. A composite score was calculated for the three emotional factors associated with anxiety and depression (HADS-Anxiety and HADS-Depression), and stress

perception (PSS) using a principal component analysis. The extracted component accounted for 74% of the variance in the three emotional scales, and all three loaded strongly on this factor (loadings ranged from 0.806 to 0.897).

Mediation and moderation effects were examined using PROCESS for SPSS (Hayes, 2013). Specifically, Model 4 with multiple parallel mediators (*negative mood, awareness and reliance on internal hunger/satiety cues*) was tested to investigate the hypothesised relationships between core symptoms of ADHD and both binge/disinhibited and restrictive eating behaviour. Indirect (i.e., mediating) effects were evaluated with 95% bias-corrected confidence intervals based on 10,000 bootstrap samples. When the confidence interval does not contain zero, this indicates that the indirect effect can be considered statistically significant (Hayes, 2013). To assess whether age, gender, BMI or ADHD medication moderates any relationship between core symptoms of ADHD and disordered eating, Model 5 was tested. Moderation is found if any of the tested interactions (e.g. inattentive symptoms of ADHD x gender) is statistically significant ($p < 0.005$). Continuous variables were mean centred as recommended by Howell (2013). All analysis was conducted using SPSS 22.0 software (IBM Corp., Armonk, NY, USA).

Covariates. Based on their potential for having associations with disordered eating and/or ADHD, it was decided a priori to include 8 variables in the statistical analyses: age, gender, BMI, socio-economic status, current ADHD medication use, other psychological/psychiatric comorbidities, alcohol and drug use. A proxy measure of socio-economic status (SES) was calculated based on the participants' responses about their highest education level completed and their current profession. Finally, alcohol and drug abuse were coded as continuous variables.

4.3.2 Results

Participants

A total of 265 individuals completed the online survey. Nine participants (3.4%) categorised themselves as “other” gender, and due to the small representation of this group in our sample population, these individuals were excluded from analyses. Further, 19 participants were excluded because of: (1) failure to meet inclusion criteria ($n = 1$; aged > 60 years old); (2) non-existent or very incomplete responses ($\geq 80\%$ of missing data) ($n = 11$); or (3) low quality responses, based on self-report measure of study engagement ($n=7$). The final sample consisted of 237 individuals.

Table 2 presents participant characteristics of the sample. Age ranged between 18 to 60 years old. Most of the participants were females (72.6%) and were of White/White British (78.1%) ethnicity. The majority of the participants were European (57.4%) or North American (35.0%) residents. More than half of the participants were highly educated (55.3%); 30.4% were university or college graduates, 23.6% had completed a Master’s degree, postgraduate certificate or diploma and 1.3% had a professional or doctoral degree. Most of the respondents were students (59.5%). A wide range of BMIs (12.49-68.52 kg/m²) was represented, with a mean of 24.6 kg/m² (SE = 0.4). Many the participants (53.6%) had a BMI within the normal range; 53 (22.4%) were classified as overweight; 32 (12.5%) as obese and 21 (8.9%) as underweight. Four participants had missing data and therefore their BMI could not be calculated.

Table 2*Participant Characteristics of the Study 1 Sample*

Total sample (n = 237)				
Demographics	Mean	SE	Min	Max
Age (years)	26.8	0.6	18	60
Gender	n	%		
Female	172	72.6		
Male	65	27.4		
Ethnic Background	n	%		
White/White British	185	78.1		
Asian/Asian British	24	10.1		
Black/African/Caribbean/Black British	4	1.7		
Mixed/multiple ethnic groups	10	4.2		
Other ethnic group	14	5.9		
Country of Residence	n	%		
UK	109	46		
Other Europe	27	11.4		
North America	83	35.0		
South America	1	0.4		
Australia/New Zealand	8	3.4		
Asia	7	3.0		
Other	2	0.8		
Education (<i>highest education completed</i>)	n	%		
Some secondary or high school education (including O Levels and GCSEs)	7	3.0		

A Levels, High School Diploma, High School Certificate, etc.	73	30.8		
Vocational qualifications	13	5.5		
University or college graduate (e.g. Bachelor's degree)	72	30.4		
Master's degree, post-graduate certificate or diploma	56	23.6		
Professional or doctoral degree (e.g. MD, PhD)	3	1.3		
Other (please specify)	13	5.5		
Profession status	n	%		
Higher managerial, administrative or professional	5	2.1		
Intermediate managerial, administrative or professional	26	11.0		
Supervisor or clerical, junior managerial, administrative or professional	23	9.7		
Skilled manual worker	3	1.3		
Semi-skilled or unskilled manual worker	8	3.4		
Student	138	58.2		
Unemployed	18	7.6		
Other (student)	16 (3)	6.8 (1.3)		
Weight	Mean	SE	Min	Max
BMI (kg/m ²)	24.6	0.4	12.5	68.5
Weight category	n	%		
Underweight	21	8.9		
Normal weight	127	53.6		
Overweight	53	22.4		
Obese	32	13.5		

Data Quality

The vast majority of the responders (97.5%) answered all of the 3 trap questions correctly, suggesting that they were paying close attention to directions. Only 2 participants (0.8%) responded correctly to 1 out of 3 questions.

Current ADHD Symptoms

ADHD symptom severity varied across participants. Table 3 presents the mean and SE, as well as the minimum (min) and the maximum (max) scores for the 4 subscales derived from the CAARS: S-SV. Of the 237 individuals, 79 (males = 20; females = 59) had received a diagnosis of ADHD, and 59 were currently being treated with medication (stimulant = 50; non-stimulant = 9). The mean reported age of ADHD diagnosis was 20.06 years (SE = 1.18); 35.4% reported being diagnosed before the age of 18 years and 64.6% reported being diagnosed in adult life.

Table 3

Mean, SE, Min and Max Scores on the Four Subscales Derived from the CAARS: S-SV

Total sample (n = 237)				
Current ADHD Symptoms	Mean	SE	Min	Max
Inattentive Symptoms (0-27)	13.80	0.50	1	27
Hyperactive-Impulsive Symptoms (0-27)	10.18	0.41	0	27
Total ADHD Symptoms (0-54)	23.97	0.85	3	54
ADHD Index (0-36)	16.67	0.52	3	34

Multiple Mediation Analyses

The path coefficients, standard errors, and other statistics pertinent to the models are superimposed on the statistical diagrams in Figures 1 and 2. Age, gender, BMI, SES, other

comorbid psychiatric disorders, current ADHD medication use, and alcohol use were covariates in the models. Drug use was found to be significantly correlated with alcohol use ($r = 0.33$), and therefore was not included as an additional covariate.

Inattentive symptoms of ADHD predicted binge/disinhibited eating both directly and indirectly. As shown in Figure 1, increased inattentive symptoms of ADHD predicted increased negative mood symptoms ($a_1 = 0.931$), and increased negative mood symptoms predicted higher binge/disinhibited eating ($b_1 = 0.284$). A bias-corrected bootstrap confidence interval for the indirect effect ($a_1b_1 = 0.264$) based on 10,000 bootstrap samples was above zero (0.114 to 0.456). Inattentive symptoms of ADHD also predicted binge/disinhibited eating indirectly through an effect on awareness and reliance on internal hunger/satiety cues, $a_2b_2 = 0.357$ BCa CI [0.157, 0.602]. Increased inattentive symptoms of ADHD predicted lower levels of awareness and reliance on internal hunger/satiety cues ($a_2 = -0.028$), and lower levels of awareness and reliance on internal hunger/satiety cues predicted higher binge/disinhibited eating ($b_2 = -12.609$). The mediating effect of negative mood was neither higher nor lower than the mediating effect of awareness and reliance on internal hunger/satiety cues BCa CI [-0.378, 0.162]. There was also evidence that inattentive symptoms of ADHD influenced binge/disinhibited eating independent of an effect on mood and awareness and reliance on internal hunger/satiety cues ($c' = 0.359$, $p = 0.041$).

Inattentive symptoms of ADHD also predicted restrictive eating, but only indirectly. As shown in Figure 1, increased inattentive symptoms predicted increased negative mood symptoms ($a_1 = 0.931$), and increased negative mood symptoms predicted higher levels of restrictive eating ($b_1 = 0.240$). A bias-corrected bootstrap confidence interval for the indirect effect ($a_1b_1 = 0.223$) based on 10,000 bootstrap samples was above zero (0.130 to 0.345). Inattentive symptoms of ADHD also predicted restrictive eating indirectly through an effect

on awareness and reliance on internal hunger/satiety cues, $a_2b_2 = 0.077$ BCa CI [0.022, 0.178]. Increased inattentive symptoms of ADHD predicted lower levels of awareness and reliance on internal hunger/satiety cues ($a_2 = -0.028$), and lower levels of awareness and reliance on internal hunger/satiety cues predicted higher restrictive eating ($b_2 = -2.736$). The mediating effect of negative mood was stronger than the mediating effect of awareness and reliance on internal hunger/satiety cues BCa CI [0.023, 0.278]. There was no evidence that inattentive symptoms of ADHD influenced restrictive eating independent of an effect on mood and awareness and reliance on internal hunger/satiety cues ($c' = 0.014$, $p = 0.893$).

Hyperactive/impulsive symptoms of ADHD also indirectly predicted binge/disinhibited eating. As shown in Figure 2, increased hyperactive/impulsive symptoms predicted increased negative mood symptoms ($a_1 = 0.574$), and increased negative mood symptoms predicted higher levels of binge/disinhibited eating ($b_1 = 0.320$). A bias-corrected bootstrap confidence interval for the indirect effect ($a_1b_1 = 0.184$) based on 10,000 bootstrap samples was above zero (0.066 to 0.351). While decreased awareness and reliance on internal hunger/satiety cues predicted higher binge/disinhibited eating ($b_2 = -12.801$), awareness and reliance on internal hunger/satiety cues was not a significant mediator as it did not relate to hyperactive/impulsive symptoms of ADHD ($p > 0.05$). There was no evidence that hyperactive/impulsive symptoms of ADHD influenced binge/disinhibited eating independent of an effect on mood ($c' = 0.270$, $p = 0.128$).

Hyperactive/impulsive symptoms of ADHD also predicted restrictive eating indirectly. As shown in Figure 2, increased hyperactive/impulsive symptoms predicted increased negative mood symptoms ($a_1 = 0.574$), and increased negative mood symptoms predicted higher levels of restrictive eating ($b_1 = 0.226$). A bias-corrected bootstrap confidence interval for the indirect effect ($a_1b_1 = 0.130$) based on 10,000 bootstrap samples

was above zero (0.059 to 0.225). Decreased awareness and reliance on internal hunger/satiety cues predicted higher restrictive eating ($b_2 = -2.718$), but awareness and reliance on internal hunger/satiety cues was not a significant mediator, as it did not relate to hyperactive/impulsive symptoms of ADHD ($p > 0.05$). There was no evidence that hyperactive/impulsive symptoms of ADHD influenced restrictive eating independent of an effect on mood ($c' = 0.164$, $p = 0.111$).

Moderation analyses

Conditional path analysis indicated that none of the relationships between core symptoms of ADHD and disordered eating were moderated by age, gender, BMI or ADHD medication ($p > 0.005$).

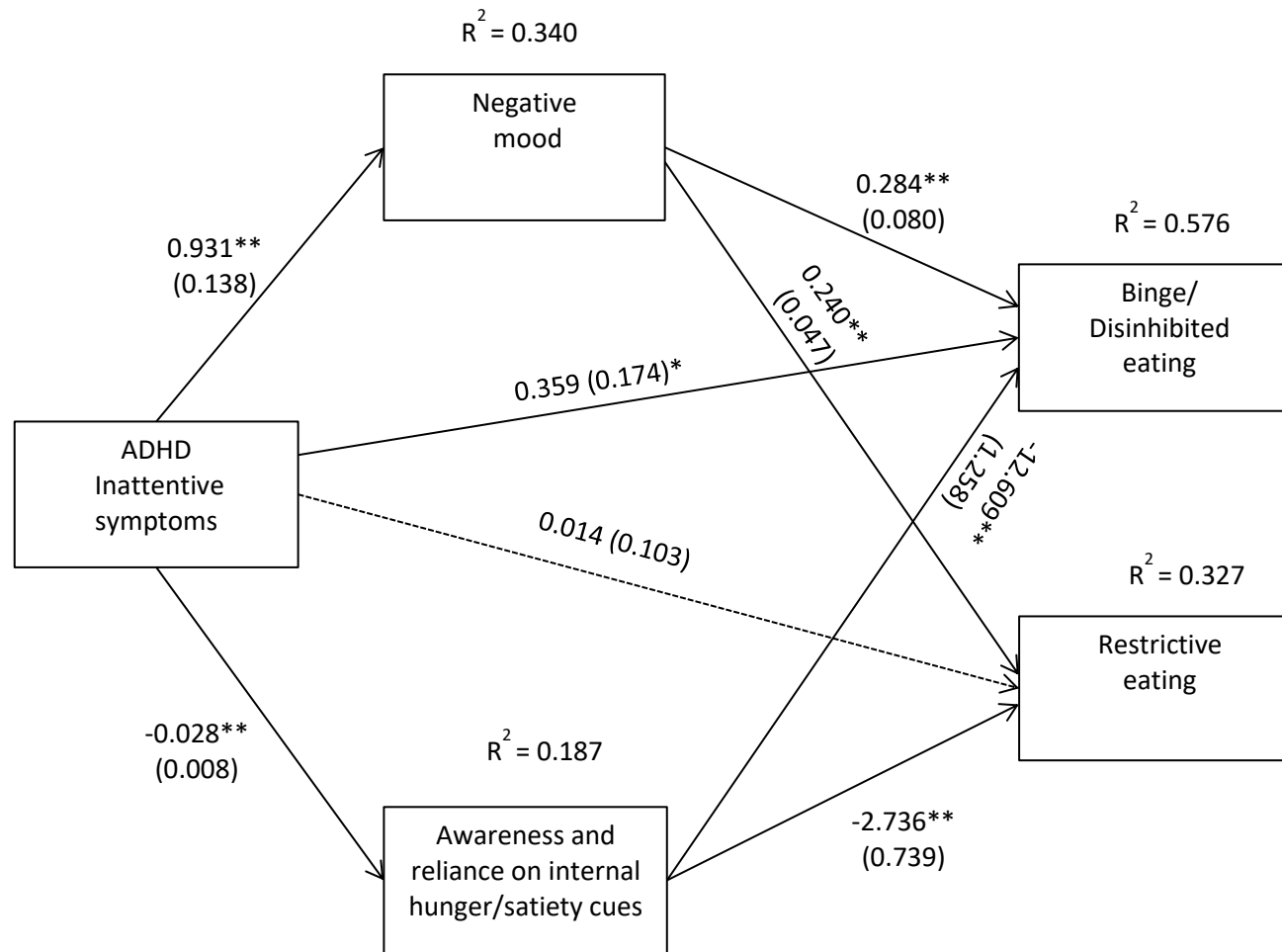


Figure 1. Statistical diagram with path coefficients and their explained variance (R^2) for the consequents. Estimates are unstandardised regression coefficients; numbers in parentheses are bootstrapped standard errors. All analyses controlled for age, gender, BMI, SES, other comorbid psychiatric disorders, current ADHD medication use, and alcohol use. Simple arrows: significant path coefficients, dotted arrows: non-significant path coefficients. * $p < 0.05$; ** $p < 0.01$

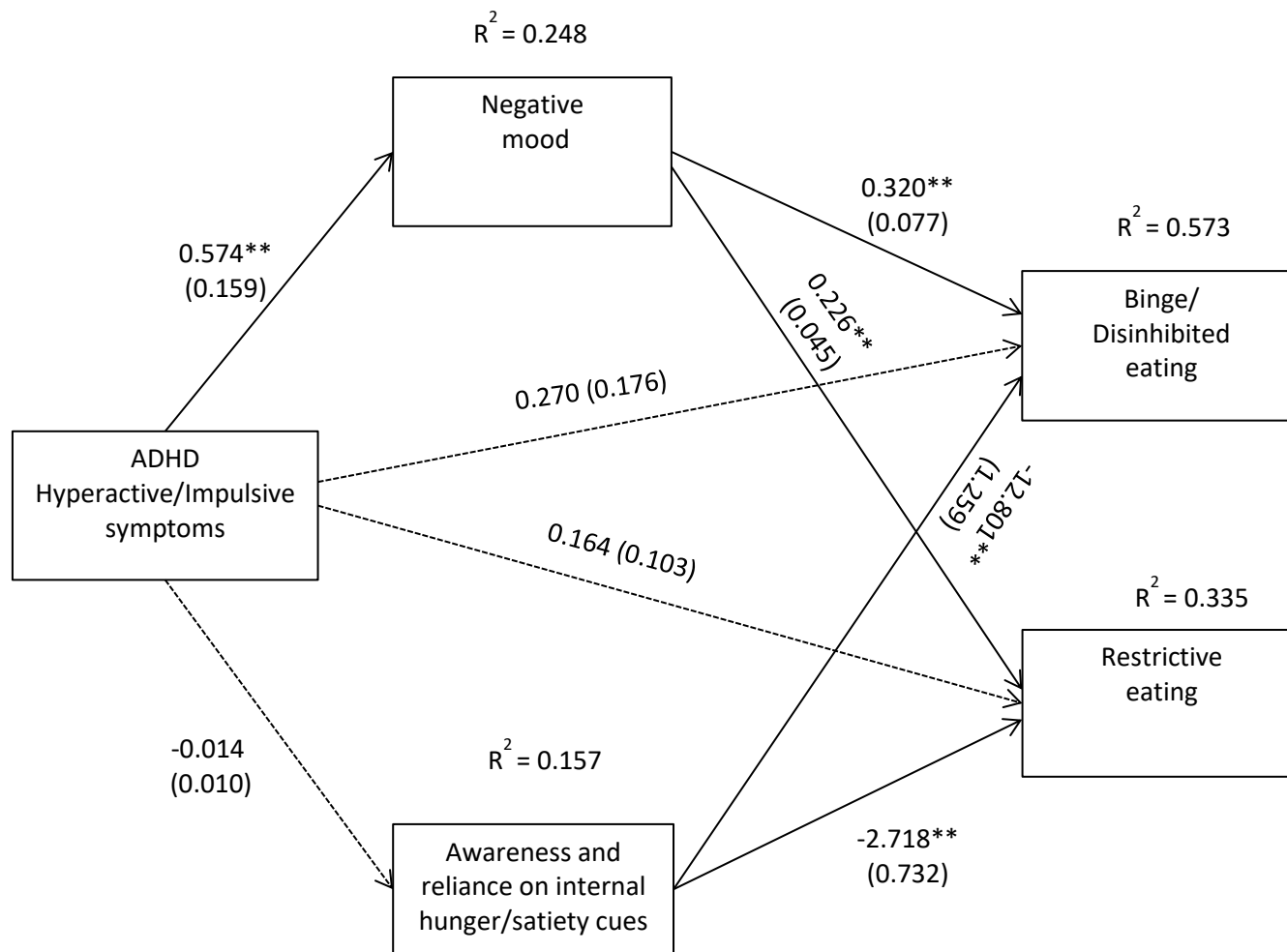


Figure 2. Statistical diagram with path coefficients and their explained variance (R^2) for the consequents. Estimates are unstandardised regression coefficients; numbers in parentheses are bootstrapped standard errors. All analyses controlled for age, gender, BMI, SES, other comorbid psychiatric disorders, current ADHD medication use, and alcohol use. Simple arrows: significant path coefficients, dotted arrows: non-significant path coefficients. * $p<0.05$; ** $p<0.01$

4.3.3 Discussion

The findings from Study 1 suggest that core symptoms of ADHD relate to binge/disinhibited and restrictive eating, both directly in the case of inattentive symptoms and binge/disinhibited eating and indirectly via a composite index of negative mood. Conditional path analysis showed that none of the relationships between core symptoms of ADHD and disordered eating were moderated by age, gender, BMI or ADHD medication. While there is strong consistent evidence for a positive association between ADHD and binge/disinhibited eating, evidence for an association between ADHD and restrictive eating has been inconsistent (for a review see Kaisari et al., 2017). Our results show for first time a direct relationship between inattentive symptoms of ADHD and binge/disinhibited eating behaviour. The findings from Study 1 also showed that inattentive, but not hyperactive/impulsive, symptoms of ADHD predicted lower levels of awareness and reliance on internal hunger/satiety cues, which in turn predicted higher disordered eating, providing another pathway of association between ADHD symptomatology and disordered eating. To the best of our knowledge, this is the first study to assess the potential mediating effect of awareness and/or reliance on internal hunger and satiety signals.

4.4 Study 2

The aim of Study 2 was to investigate whether the online findings from Study 1 could be replicated in a well-defined clinical laboratory sample. In Study 2, we also investigated the specific contributions (*if any*) of hyperactivity and impulsivity to disordered eating. As the derived subscales of the CAARS-S:SV, do not provide separate scores for hyperactive and impulsive symptoms of ADHD, it is unclear whether both hyperactive and impulsive symptoms relate to binge/disinhibited and restrictive eating or specific symptoms relate to specific types of disordered eating. In a systematic review of the literature (Kaisari et al.,

2017), we found that impulsivity symptoms of ADHD were positively related to bulimic behaviour. However, no association was found between hyperactivity symptoms of ADHD and bulimic behaviour. Interestingly, hyperactivity symptoms were positively related to restrictive eating (*e.g. drive for thinness, dietary restraint*), particularly in men (Grabarek & Cooper, 2008; Slane, Burt, & Klump, 2010). However, given that only two studies to date have assessed the relationship between hyperactivity symptoms of ADHD and restrictive eating in adults, further research is required to replicate these initial findings. In Study 2, we investigated the specific contributions (*if any*) of hyperactivity and impulsivity to disordered eating via assessment of impulsivity both by a self-report measure and a performance-based measure. We hypothesised that both measures of impulsivity would correlate positively with the hyperactive/impulsive symptoms subscale of the CAARS-S:SV. In addition, we hypothesised that if impulsive symptoms of ADHD relate mainly to binge/disinhibited eating and hyperactive symptoms to restrictive eating, a similar pattern of results would be observed when measures of impulsivity (*instead of the hyperactive/impulsive symptoms subscale of the CAARS-S:SV*) were used as predictors of binge/disinhibited eating behaviour, while a different pattern would be expected when impulsivity measures were used as predictors of restrictive eating behaviour.

In Study 2, we assessed awareness and reliance on internal hunger/satiety cues via a self-report measure, as in Study 1, but additionally used a performance-based measure of interoceptive sensitivity, heartbeat perception. The heartbeat perception task has been related to the ability to detect changes in other autonomically innervated organs, such as the activity of the stomach (Herbert, Muth, Pollatos, & Herbert, 2012; Whitehead & Drescher, 1980), highlighting its role as an indicator of a generalised sensitivity for visceral processes in situations evoking interoceptive signals (Herbert & Pollatos, 2012), even during food

deprivation and feeling hungry (Herbert, Herbert, et al., 2012). We hypothesised that inattentive symptoms of ADHD would relate negatively to interoceptive sensitivity, and decreased interoceptive sensitivity would relate positively to disordered eating.

Overall, Study 2 aimed to: (i) investigate whether the findings from Study 1 can be replicated using a well-defined laboratory sample, (ii) investigate the specific contributions (*if any*) of hyperactivity and impulsivity to disordered eating, via assessment of impulsivity both by a self-report measure and a performance-based measure, and (iii) investigate whether interoceptive sensitivity as assessed objectively via a heartbeat perception task provides another pathway of association between inattentive symptoms of ADHD and disordered eating as found in Study 1, when we used a self-report measure of awareness and reliance on internal hunger/satiety cues.

4.4.1 Materials and Methods

Participants and Procedure

Students from the University of Birmingham, UK were recruited in exchange for course credits or cash payment. From October 2016 to December 2016, a total 142 individuals completed the study. Participants were required to be fluent in English. The study was advertised as a two-part study, and participants had to complete an online questionnaire before a lab meeting was arranged to complete the second part of the study and receive full reimbursement for their participation. All participants provided online informed consent before completion of the online questionnaire. After informed consent was obtained, participants were directed to a URL to complete an online survey as described in Study 1. Following completion of the survey, a lab meeting was arranged and participants came to the School of Psychology, University of Birmingham to complete a battery of behavioural measures. Informed consent was also obtained before completion of the behavioural

measures. The protocol was approved by the University of Birmingham Research Ethics Committee.

Measures

Data quality. Trap questions and a self-report measure of study engagement were used to enable a quality assessment of the collected responses, as described for Study 1.

Demographics

Demographics including age, sex, ethnicity, education level and professional status were reported as in Study 1. Participants height and body weight were measured to calculate current BMI (kg/m^2).

Questionnaire Measures

Questionnaire measures for disordered eating, awareness and reliance on internal hunger/satiety cues, negative mood, alcohol and drug use were the same as in Study 1. The Cronbach's alpha values in the present study are summarised in Table 4.

Table 4*Cronbach's Alpha Values for the Questionnaire Measures in Study 2*

Measures	Cronbach's alpha
<i>Disordered Eating</i>	
DEBQ - Emotional eating	0.96
DEBQ - External eating	0.86
DEBQ - Dietary Restraint	0.94
LOCES	0.93
BES	0.91
BITE	0.89
EAT Total score	0.91
EAT - Dieting	0.88
EAT - Bulimia	0.86
EAT - Oral Control	0.68
<i>Awareness and reliance on internal hunger/satiety cues</i>	
RIH	0.78
<i>Negative mood</i>	
HADS - Anxiety	0.82
HADS - Depression	0.75
PSS	0.87
<i>Covariates</i>	
SMAST [£]	0.51
DAST-10	0.46

[£] Based on total 9 scale items. Items 8, 10, 12 and 13 had zero variance and were removed.

Impulsivity

The Barratt Impulsivity Scale (BIS) (Patton et al., 1995), a 30-item self-report questionnaire, was used in Study 2 to assess impulsivity. The BIS utilises a 4-point format in which respondents are asked to rate items pertaining to impulsive or non-impulsive (for reverse scored items) behaviours and preferences. The BIS identifies three aspects of impulsivity: (1) inability to concentrate and focus attention (*attention impulsiveness*), (2) acting without thinking (*motor impulsiveness*) and (3) lack of future planning (*non-planning impulsiveness*) (Stanford et al., 2009). Currently, this is the most widely used self-report scale of trait impulsivity; its psychometric properties have been determined in both clinical and non-clinical subjects (Patton, Stanford, & Barratt, 1995; Stanford et al., 2009). In the present study, the Cronbach's alpha was 0.85, 0.76, 0.67 and 0.68 for the total score, attention impulsiveness, motor impulsiveness and non-planning impulsiveness, respectively.

Go/No-Go Task. Impulsivity was also assessed behaviourally using a go/no-go task assessing the ability to inhibit instigated, "prepotent" responses (*response inhibition*). Participants were instructed to press the space bar in response to explicitly instructed 'go' stimuli and withhold any response to 'no-go' stimuli. Participants completed two tasks: (a) a neutral go/no-go task and (b) a food go/no go task. Both the neutral and food-based go/no-go tasks were programmed using E-Prime 2.0 software (Psychology Software tools, Inc., Sharpsburg, PA, USA; see Figure 3). In the neutral task, sports equipment and toiletries images were used; there were two conditions, with the sports equipment images comprising the "go" stimuli in one condition (toiletries images: "no go" stimuli) and the "no go" stimuli in the other condition (toiletries images: "go" stimuli).

In the food-based task, high-energy dense food and low-energy dense food images were used. Similarly, to the neutral task there were two conditions, with the high-energy

dense food images comprising the ‘go’ stimuli in one condition (low-energy dense food images: ‘no go’ stimuli) and the ‘no go’ stimuli in the other condition (low-energy dense food images: ‘go’ stimuli). The stimuli were selected from the BOSS database of normative photographs of objects (Brodeur, Dionne-Dostie, Montreuil, & Lepage, 2010). The BOSS images are normalised for category, familiarity, visual complexity, object agreement and viewpoint agreement, making them equivalent in valence and perceptual characteristics (Brodeur et al., 2010). All participants completed both tasks. The four conditions were presented in a randomised order. The measure of interest was the number of commission errors (responses incorrectly made in ‘no-go’ trials). In the present study, the total number of commission errors for the neutral task and the food task was the main outcome of interest.

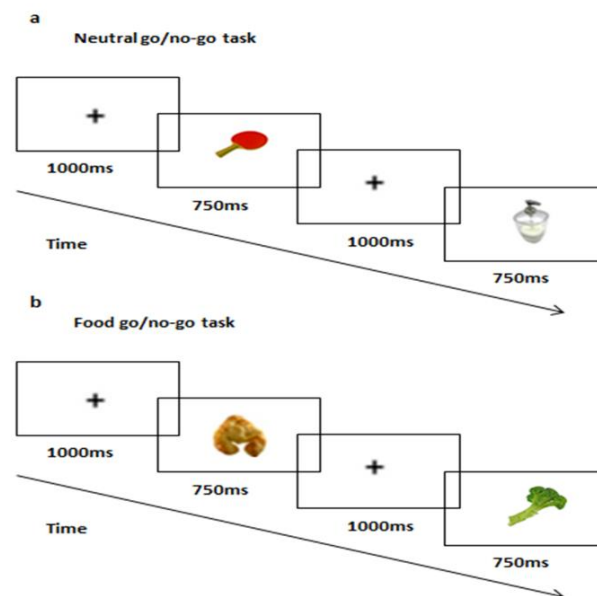


Figure 3. Go/no-go task: A total of 320 trials were presented, 160 (in two blocks of 80; 20 trials/condition) for the neutral task (a) and the same for the food task (b). Images were presented using a ratio of 80% ‘go’ to 20% ‘no-go’ trials to create a prepotent ‘go’ response. Each trial was presented for 750 ms and was preceded by a fixation point for 1000 ms. The go and no-go categories were presented in a randomised order. Presentation order of the food and neutral tasks was randomised.

Interoceptive sensitivity: the heartbeat perception task. Interoceptive sensitivity was assessed using the heartbeat perception task. For the heartbeat task, participants' heart rate was monitored with a pulse oximeter (PulseOximeterOnline.com) to obtain their average heart rate. Following the well-validated Mental Tracking Method of Schandry (1981), data were recorded for two-time intervals (45 s, 60 s). While participants' heart rate was monitored with a pulse oximeter, participants were asked to count silently their own heartbeats, without taking their pulse or attempting any other physical manipulation that could facilitate the detection of heartbeats. Participants were given verbal instructions when to start counting and when to stop counting. At the end of each time interval, participants were asked to verbally report how many heartbeats they counted. No feedback on the duration of the counting phases or the quality of their performance was given. Interoceptive sensitivity was measured using a heartbeat perception score, calculated as the mean score across two heartbeat perception intervals according to the following transformation: $\frac{1}{2} \sum (1 - (|\text{recorded heartbeats} - \text{counted heartbeats}|) / \text{recorded heartbeats})$. The heartbeat perception score varies between 0 and 1. The maximum score of 1 indicates absolute accuracy of heartbeat perception.

Analysis Plan

Data reduction.

Disordered eating. As in Study 1, composite scores were created for “Binge/Disinhibited Eating” for “Restrictive Eating”.

Negative mood. A composite score was calculated as in Study 1. In the current study, the extracted component accounted for 77% of the variance in the three emotional scales, and all three scales loaded strongly on this factor (range from 0.855 to 0.893).

As in Study 1, it was decided a priori that responses of participants who answered all three trap questions incorrectly and responses of participants who responded negatively to the self-report measure of study engagement would be removed. Mediation and moderation

effects were examined using PROCESS for SPSS (Hayes, 2013) (for details see Analysis Plan Study 1). To investigate whether both measures of impulsivity (*BIS; commission errors on the go/no go task*) correlated positively with the hyperactivity/impulsivity symptoms subscale of the CAARS-S:SV as hypothesised, Spearman's correlations for non-normally distributed variables were computed. Spearman's correlations were also computed to assess whether inattentive symptoms of ADHD correlated negatively to interoceptive sensitivity, and decreased interoceptive sensitivity correlated positively to disordered eating.

Covariates. As in Study 1, the following variables were included in the statistical analyses: age, gender and BMI. Current ADHD medication use and other psychological/psychiatric comorbidities were not included in the statistical analyses, as none of the participants reported either use of ADHD medication or any psychological/psychiatric comorbidity. In addition, SES was not included in the statistical analysis, as all participants were students. The internal consistency for the SMAST questionnaire was poor in the present study (Cronbach's alpha = 0.51). In addition, only 7 (4.9%) participants in total reported a score equal to or greater than 3 which has been suggested to indicate a borderline alcohol problem or potential alcohol abuse. Therefore, alcohol use was not included as a covariate in the statistical analysis.

4.4.2 Results

Participants

Table 5 presents participant characteristics of the Study 2 sample. A total 142 participants completed the study. Age ranged from 18 to 32 years old. Most of the participants were females (80.3%) and were of White/White British (68.3%) ethnicity. BMIs ranged between 14.7-34.4 kg/m², with a mean of 21.4 kg/m² (SE = 0.3). The majority of the participants (65.5%) had a BMI within the normal range; 26 (18.3%) were classified as

underweight; 20 (14.1%) as overweight and 2 (1.4%) as obese. One participant had missing data and therefore their BMI could not be calculated.

Table 5

Participant Characteristics of the Study 2 Sample

Total sample (n = 142)				
Demographics	Mean	SE	Min	Max
Age (years)	19.3	0.1	18	32
Gender	n	%		
Female	114	80.3		
Male	28	19.7		
Ethnic Background	n	%		
White/White British	97	68.3		
Asian/Asian British	27	19.0		
Black/African/Caribbean/Black British	10	7.0		
Mixed/multiple ethnic groups	4	2.8		
Other ethnic group	4	2.8		
Weight	Mean	SE	Min	Max
BMI (kg/m ²)	21.4	0.3	14.7	34.4
Weight category	n	%		
Underweight	26	18.3		
Normal weight	93	65.5		
Overweight	20	14.1		
Obese	2	1.4		

Data Quality

The vast majority of the responders (98.6%) answered all of the 3 trap questions correctly, suggesting that they were paying close attention to directions.

Current ADHD Symptoms

Table 6 presents the mean and SE, as well as the minimum (min) and the maximum (max) scores for the 4 subscales derived from the CAARS: S-SV.

Table 6

Mean, SE, Min and Max Scores on the Four Subscales Derived from the CAARS: S-SV

Total sample (n = 142)				
Current ADHD Symptoms	Mean	SE	Min	Max
Inattentive Symptoms (0-27)	9.23	0.43	0	24
Hyperactive-Impulsive Symptoms (0-27)	7.25	0.40	0	22
Total ADHD Symptoms (0-54)	16.49	0.75	0	45
ADHD Index (0-36)	11.71	0.47	2	31

Multiple Mediation Analyses

Aim 1: To investigate whether the findings from Study 1 can be replicated using a well-defined laboratory sample.

The path coefficients, standard errors, and other statistics pertinent to the models are superimposed on the statistical diagrams in Figures 4 and 5.

Inattentive symptoms of ADHD predicted binge/disinhibited eating both directly and indirectly. As shown in Figure 4, increased inattentive symptoms of ADHD predicted increased negative mood symptoms ($a_1 = 1.273$), and increased negative mood symptoms predicted higher binge/disinhibited eating ($b_1 = 0.472$). A bias-corrected bootstrap confidence

interval for the indirect effect ($a_1b_1 = 0.600$) based on 10,000 bootstrap samples was above zero (0.299 to 1.013). Inattentive symptoms of ADHD also predicted binge/disinhibited eating indirectly through an effect on awareness and reliance on internal hunger/satiety cues, $a_2b_2 = 0.227$ BCa CI [0.012, 0.544]. Increased inattentive symptoms of ADHD predicted lower levels of awareness and reliance on internal hunger/satiety cues ($a_2 = -0.021$), and lower levels of awareness and reliance on internal hunger/satiety cues predicted higher binge/disinhibited eating ($b_2 = -10.581$). The mediating effect of negative mood was neither higher nor lower than the mediating effect of awareness and reliance on internal hunger/satiety cues BCa CI [-0.016, 0.770]. There was also evidence that inattentive symptoms of ADHD influenced binge/disinhibited eating independent of an effect on mood and awareness and reliance on internal hunger/satiety cues ($c' = 0.666$, $p = 0.012$).

Inattentive symptoms of ADHD predicted also restrictive eating, but only indirectly. As shown in Figure 4, increased inattentive symptoms predicted increased negative mood symptoms ($a_1 = 1.273$), and increased negative mood symptoms predicted higher levels of restrictive eating ($b_1 = 0.217$). A bias-corrected bootstrap confidence interval for the indirect effect ($a_1b_1 = 0.276$) based on 10,000 bootstrap samples was above zero (0.117 to 0.490). While, increased inattentive symptoms of ADHD predicted lower levels of awareness and reliance on internal hunger/satiety cues ($a_2 = -0.021$), and lower levels of awareness and reliance on internal hunger/satiety cues predicted higher restrictive eating ($b_2 = -3.066$), a bias-corrected bootstrap confidence interval for the indirect effect ($a_1b_1 = 0.066$) based on 10,000 bootstrap samples was below zero (-0.003 to 0.227), suggesting that the mediating effect was not significant. There was no evidence that inattentive symptoms of ADHD influenced restrictive eating independent of an effect on mood and awareness and reliance on internal hunger/satiety cues ($c' = 0.250$, $p = 0.115$).

Hyperactive/impulsive symptoms of ADHD predicted binge/disinhibited eating both directly and indirectly. As shown in Figure 5, increased hyperactive/impulsive symptoms predicted increased negative mood symptoms ($a_1 = 1.024$), and increased negative mood symptoms predicted higher levels of binge/disinhibited eating ($b_1 = 0.467$). A bias-corrected bootstrap confidence interval for the indirect effect ($a_1b_1 = 0.478$) based on 10,000 bootstrap samples was above zero (0.243 to 0.848). While decreased awareness and reliance on internal hunger/satiety cues predicted higher binge/disinhibited eating ($b_2 = -11.426$), awareness and reliance on internal hunger/satiety cues was not a significant mediator as it did not relate to hyperactive/impulsive symptoms of ADHD ($p > 0.05$). There was also evidence that hyperactive/impulsive symptoms of ADHD influenced binge/disinhibited eating independent of an effect on mood ($c' = 0.939$, $p < 0.01$).

Hyperactive/impulsive symptoms of ADHD predicted restrictive eating both directly and indirectly. As shown in Figure 5, increased hyperactive/impulsive symptoms predicted increased negative mood symptoms ($a_1 = 1.024$), and increased negative mood symptoms predicted higher levels of restrictive eating ($b_1 = 0.218$). A bias-corrected bootstrap confidence interval for the indirect effect ($a_1b_1 = 0.223$) based on 10,000 bootstrap samples was above zero (0.110 to 0.406). Decreased awareness and reliance on internal hunger/satiety cues predicted higher restrictive eating ($b_2 = -3.371$), but awareness and reliance on internal hunger/satiety cues was not a significant mediator, as it did not relate to hyperactive/impulsive symptoms of ADHD ($p > 0.05$). There was also evidence that hyperactive/impulsive symptoms of ADHD influenced restrictive eating independent of an effect on mood ($c' = 0.334$, $p = 0.036$).

Moderation Analyses

Conditional path analysis indicated that none of the relationships between core symptoms of ADHD and disordered eating were moderated by age, gender, or BMI ($p_s > 0.005$).

Aim 2: To investigate the specific contributions (*if any*) of hyperactivity and impulsivity to disordered eating, via assessment of impulsivity both by a self-report measure and by a performance-based measure.

As hypothesised, hyperactivity/impulsivity symptoms of ADHD, assessed by the Hyperactive-impulsive subscale of the CAARS-S: SV, were positively correlated with self-reported levels of impulsivity ($r = 0.54$; $p < 0.001$), assessed by the BIS. However, contrary to our hypothesis, hyperactivity/impulsivity symptoms of ADHD, were not correlated with either the number of commission errors on the neutral no-go trials ($r = 0.13$; $p_s = 0.13$) or the food no-go trials ($r = 0.14$; $p = 0.11$). Therefore, to investigate the specific contributions (*if any*) of hyperactivity and impulsivity to disordered eating, we tested the model shown in Figure 5 using impulsivity assessed by the BIS as the predictor variable.

The path coefficients, standard errors, and other statistics pertinent to the model are superimposed on the statistical diagram in Figure 6. Age, gender and BMI were covariates in the models. As shown in Figure 6, a similar pattern of results was observed when impulsivity assessed by the BIS was used as a predictor variable. However, although there was evidence that impulsivity influenced binge/disinhibited eating independent of an effect on mood ($c' = 0.242$, $p = 0.035$), there was no evidence of a direct effect of impulsivity on restrictive eating ($c' = -0.007$, $p = 0.915$), and by implication providing evidence that the relationship between hyperactivity/impulsivity symptoms of ADHD and restrictive eating is driven mainly by hyperactivity and not impulsivity symptoms. Investigation of the specific facets of impulsivity

showed that only attentional and motor impulsivity influenced binge/disinhibited eating independent of an effect on mood ($c' = 0.657$, $p=0.034$; $c' = 0.699$, $p=0.001$, respectively), and there was no evidence of a direct effect of non-planning impulsivity on binge/disinhibited eating ($c' = 0.140$, $p=0.602$). There was no evidence of a direct effect of any of the different components of impulsivity on restrictive eating (all $p_s > 0.005$).

Aim 3: To investigate whether interoceptive sensitivity assessed objectively by a heartbeat task mediates the association between inattentive symptoms of ADHD and disordered eating.

The mean score of interoceptive sensitivity was 0.71 ($SE = 0.02$). One participant performed poorly on the heartbeat task compared to the other participants (score $< 3SD$ from the mean), therefore their score was removed from the subsequent analysis. There was no evidence of a significant association between inattentive symptoms of ADHD and interoceptive sensitivity ($r = 0.01$, $p_s = 0.94$), nor between interoceptive sensitivity and binge/disinhibited ($r = -0.01$, $p_s = 0.91$) or restrictive eating ($r = -0.07$, $p_s = 0.40$). Thus, no mediational analysis was conducted. Similarly, interoceptive sensitivity scores were not significantly correlated with scores on the RIH subscale of the Intuitive Eating Scale ($r = -0.02$, $p_s = 0.80$).

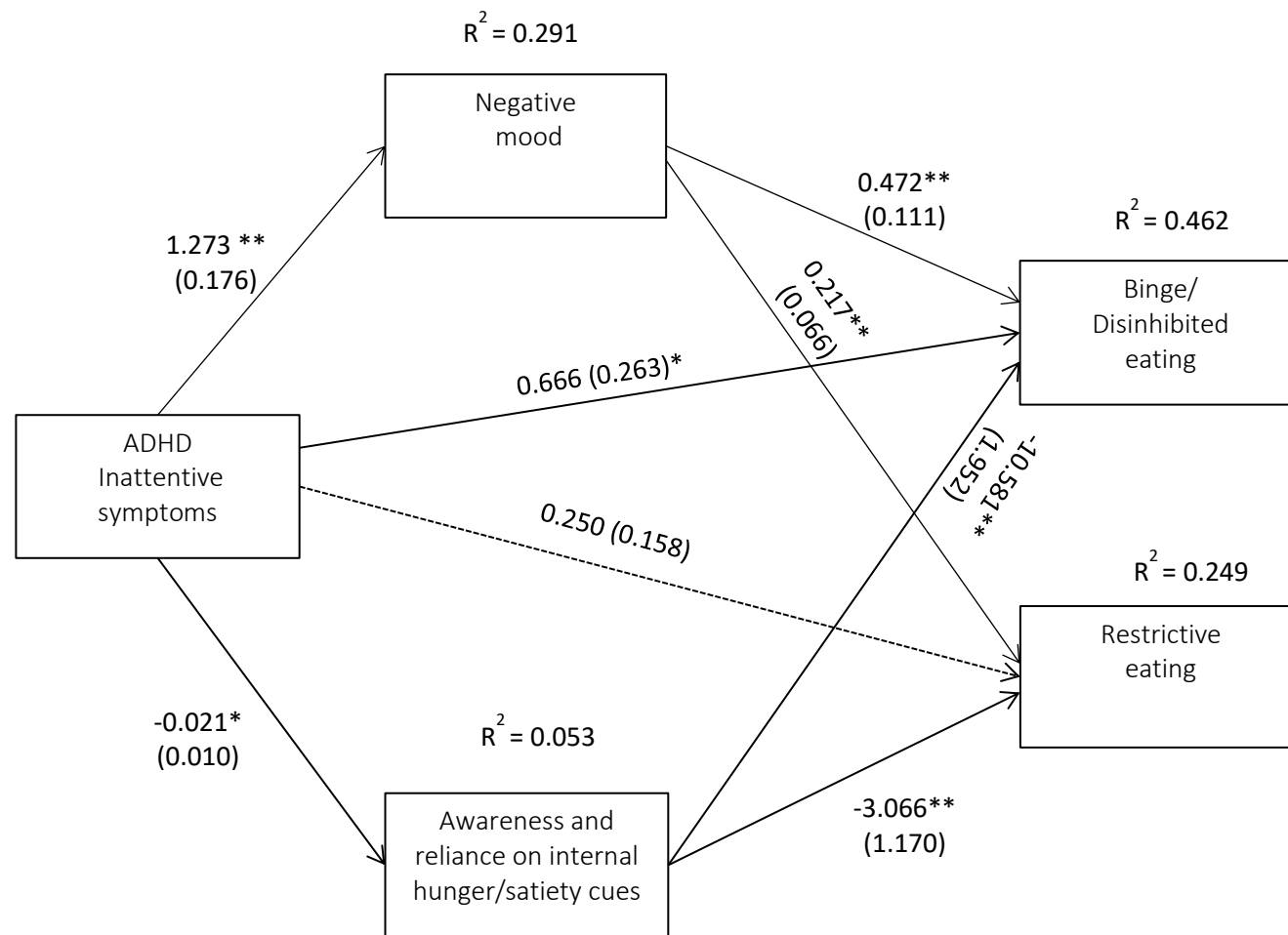


Figure 4. Statistical diagram with path coefficients and their explained variance (R^2) for the consequents. Estimates are unstandardised regression coefficients; numbers in parentheses are bootstrapped standard errors. All analyses controlled for age, gender, BMI. Simple arrows: significant path coefficients, dotted arrows: non-significant path coefficients. * $p < 0.05$; ** $p < 0.01$

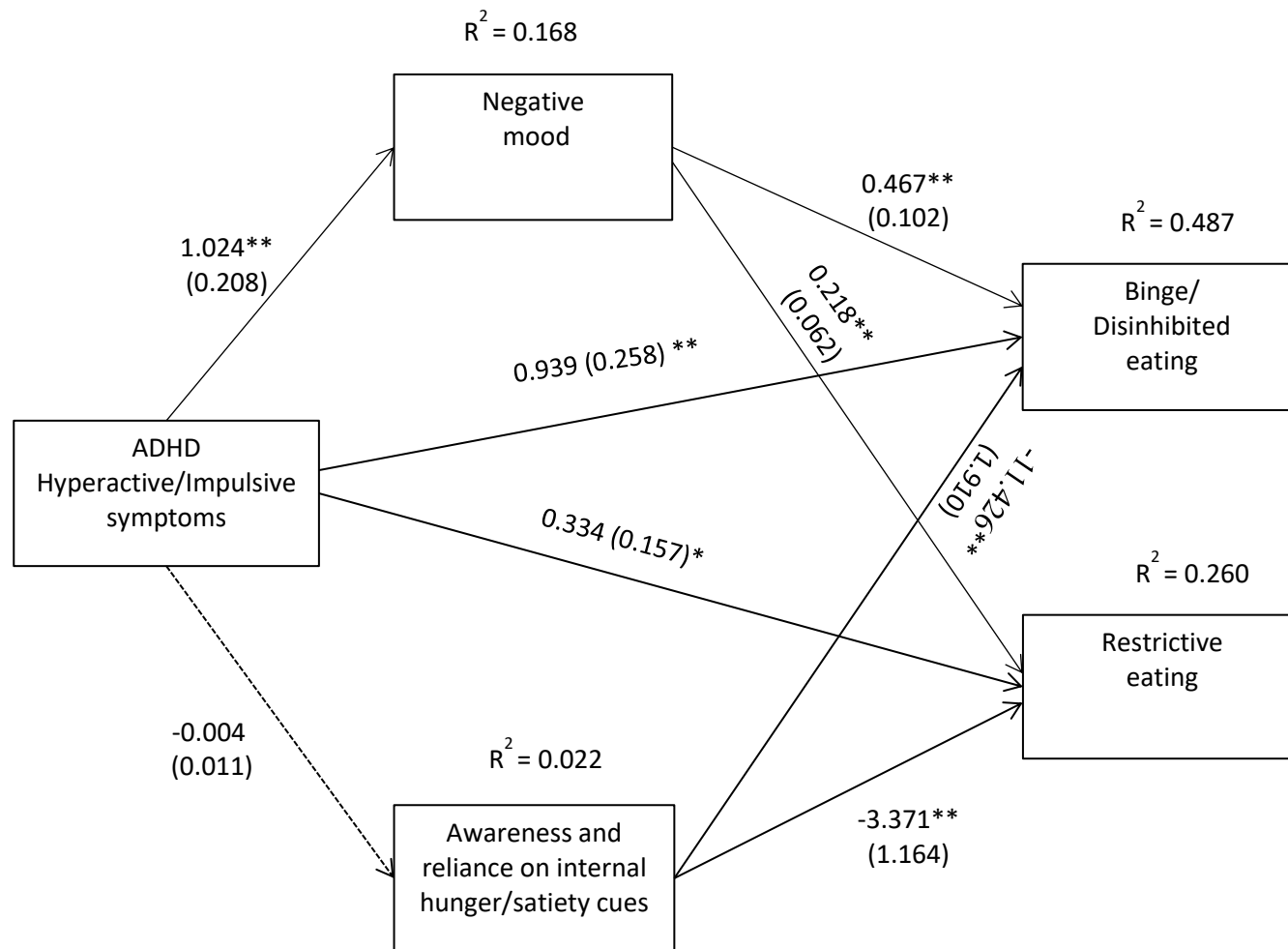


Figure 5. Statistical diagram with path coefficients and their explained variance (R^2) for the consequents. Estimates are unstandardised regression coefficients; numbers in parentheses are bootstrapped standard errors. All analyses controlled for age, gender, BMI. Simple arrows: significant path coefficients, dotted arrows: non-significant path coefficients. * $p < 0.05$; ** $p < 0.01$

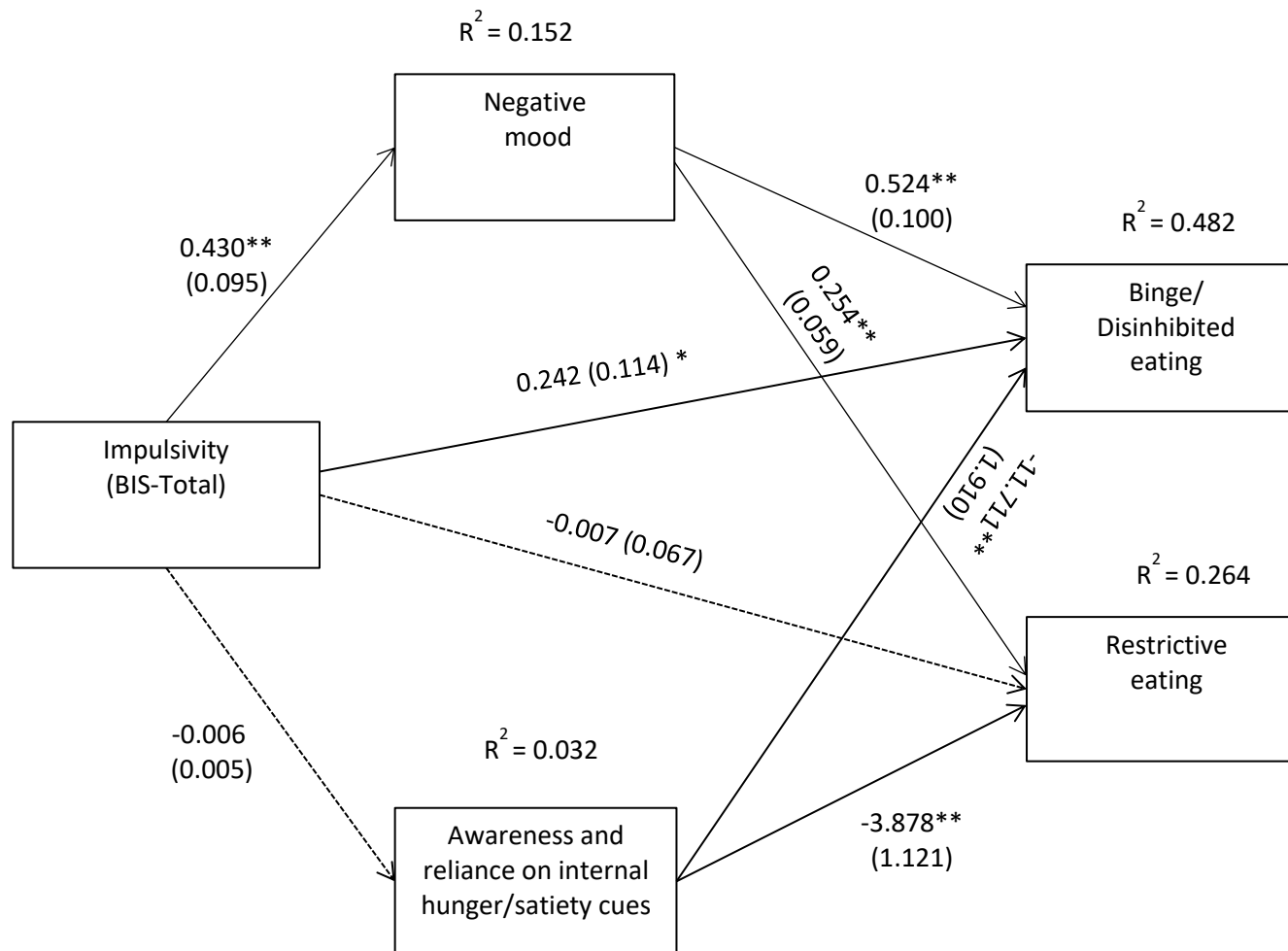


Figure 6. Statistical diagram with path coefficients and their explained variance (R^2) for the consequences. Estimates are unstandardised regression coefficients; numbers in parentheses are bootstrapped standard errors. All analyses controlled for age, gender, BMI. Simple arrows: significant path coefficients, dotted arrows: non-significant path coefficients. * $p < 0.05$; ** $p < 0.01$

4.5 Discussion

The present studies investigated associations between the core symptoms of ADHD and disordered eating, in two independent adult samples, and examined the mechanisms that may underpin these relationships. To the best of our knowledge, these are the first studies to assess both restrictive and binge/disinhibited eating behaviour associated with ADHD, and to investigate mediating effects of negative mood and deficits in awareness and reliance on internal hunger/satiety cues.

The results of the two studies demonstrated that both the inattentive and hyperactive/impulsive symptoms of ADHD were associated with both binge/disinhibited and restrictive eating. In addition, in both studies negative mood, a composite index reflecting anxiety, depression and stress was a significant mediator of the association between core symptoms of ADHD and disordered eating. Furthermore, deficits in awareness and reliance on internal hunger/satiety signals was another mediator. However inattentive, but not hyperactive/impulsive, symptoms of ADHD predicted lower levels of awareness and reliance on internal hunger/satiety cues, which in turn predicted disordered eating, especially binge/disinhibited eating. In Study 2, interoceptive sensitivity assessed by a heartbeat task was found not to mediate the relationship between inattentive symptoms of ADHD and disordered eating. The results from both studies provide the first evidence for a direct relationship between inattentive symptoms of ADHD and binge/disinhibited eating behaviour.

In Study 2, hyperactive/impulsive symptoms of ADHD were also found to directly associate with both binge/disinhibited and restrictive eating. Impulsivity directly predicted binge/disinhibited eating but there was no evidence for a direct association with restrictive eating, which by implication suggests a specific contribution of impulsivity ADHD symptoms to binge/disinhibited eating and hyperactivity symptoms to restrictive eating. Moderation

analysis showed that none of the relationships between the core symptoms of ADHD and disordered eating were influenced by age, gender, ADHD medication or BMI.

While there is strong consistent evidence for a positive association between ADHD and binge/disinhibited eating (Davis et al., 2006; Hudson, Hiripi, Pope, & Kessler, 2007; Strimas et al., 2008; Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011), an association between ADHD and restrictive eating behaviour has been rarely investigated in previous studies, and the findings have been inconsistent. For example, Swanson et al. (2011) in a nationally representative sample of 10,123 adolescents aged 13 to 18 years in the US, reported a non-significant association between ADHD and AN. However, this analysis may have been underpowered owing to the relatively small numbers of adolescents with AN ($n = 34$) in the sample. In contrast, Bleck, DeBate, and Olivardia (2015) in a recent secondary data analysis of the US National Longitudinal Study of Adolescent Health reported that participants who had been diagnosed by a health care professional with ADHD were almost 5 times more likely to report clinical level restrictive eating behaviour, defined as a combination of a previously diagnosed ED (*either AN or BN*) along with current (*past week*) restrictive behaviours. However, as participants who reported both current bingeing and/or purging and restrictive eating were included in both behaviour subtype groups, it is unclear whether the restrictive behaviours reported by participants were motivated solely by the desire to lose weight or represented a compensatory behaviour following consumption of large amounts of food. It is therefore, uncertain whether the restriction was part of a restrictive ED (e.g. AN) or of a more impulsive ED (e.g. BN or BED). In the present studies, we used a composite index of restrictive eating which reflects a pattern of behaviour to limit food intake as a means of controlling body weight, and found that ADHD symptomatology is positively associated with restrictive eating behaviour in two independent samples.

Negative mood was a significant mediational pathway of association between core symptoms of ADHD and both binge/disinhibited and restrictive eating behaviour. These findings suggest that internalising problems frequently found in individuals with ADHD may provide an underlying mechanism for disordered eating behaviours. Schweickert, Strober, and Moskowitz (1997) suggested that compulsive eating in individuals with ADHD may be a compensatory mechanism to control frustration and anxiety associated with attention and organisational difficulties. In line with this hypothesis, Yates, Lund, Johnson, Mitchell, and McKee (2009) studied a population of inpatient women with eating disorders and found that depression scores were positively associated with current ADHD inattention symptoms and Nazar et al. (2014) reported that depressive symptoms were the strongest predictor of binge eating in a sample of clinically obese women. In addition, stress and depression levels are predictive of bingeing behaviour in both individuals with an eating disorder and a student population (Greenberg, 1986; Greenberg & Harvey, 1987; Johnson, Rohan, & Kirk, 2002; Stice, 2002), suggesting that binge eating may serve as a means to control or self-regulate mood. Similarly, Abraham and Beumont (1982) reported that 72% of their subjects stated that they were free of negative mood states while binge eating. Thus, binge eating behaviour may help individuals with ADHD to self-regulate their negative mood due to the mood enhancing properties of food, especially if consumed during a binge eating episode (*when generally high fat and carbohydrate foods are consumed*) (Gibson, 2006; Guertin & Conger, 1999; Hadigan, Kissileff, & Walsh, 1989). Similarly, restrictive eating may also be used as a coping mechanism for negative affect and significant weight loss through extreme restrictive eating, as seen in AN, is another means of emotional regulation resulting in a short-term decrease in negative mood (Fairburn et al., 1999). Continued weight loss in AN can be reinforced by feelings of control and achievement, and increased self-esteem, based on a perception of low-

body weight “success” (Dignon, Beardsmore, Spain, & Kuan, 2006). Taken together these findings suggest that disordered eating, both bingeing and restrictive eating, may assist individuals with ADHD to control negative affect, especially in the absence of any other coping mechanisms. Our results extend the current state of knowledge by providing the first evidence for a mechanistic pathway of association between the core symptoms of ADHD and eating pathology via negative affect.

Decreased awareness of internal signs of hunger and satiety has been suggested as a possible pathway of association between inattentive symptoms of ADHD and disordered eating (Davis et al., 2006), but to the best of our knowledge this hypothesis has not been investigated in previous studies. The present results show that the inattentive symptoms of ADHD were associated with decreased awareness of internal signs of hunger and satiety, and in turn these deficits were positively associated with disordered eating, especially binge/disinhibited eating. Decreased awareness and/or poor knowledge of internal hunger/satiety cues could indicate that individuals with ADHD primarily use external cues to guide their eating behaviour. Avoidance of eating when hungry, in combination with decreased reliance on satiety signals, may foster overeating and explain an inability of individuals with ADHD to stop eating, especially in the presence of highly palatable foods.

We found no evidence of an association between inattentive symptoms of ADHD and interoceptive sensitivity assessed by the heart beat perception task. Similarly, we found that interoceptive sensitivity was not associated with disordered eating. Pollatos et al. (2008) used a heartbeat perception task and found that patients with AN displayed significantly decreased interoceptive sensitivity compared to controls. However, more recently Eshkevari, Rieger, Musiat, and Treasure (2014) did not find any differences in a heartbeat detection task between individuals with an eating disorder (32 with AN, 22 with BN and 20 not specified) and

healthy controls. These apparently contradictory findings suggest that further studies are required to investigate the use of a heartbeat task as an index of interoceptive sensitivity in individuals with eating disorders/disordered eating behaviours. In contrast to the report of Herbert et al. (2013) we did not find that interoceptive sensitivity correlated with the awareness and reliance on internal hunger/satiety cues subscale of the 21-item Intuitive Eating Scale (IES) (Tylka, 2006). This may suggest that the awareness and reliance on internal hunger/satiety cues subscale indexes a distinct component of interoception from the heartbeat task, at least within our sample population. Specifically, the self-report questionnaire indexes both awareness and reliance on bodily signals (*hunger/satiety*), whereas the heartbeat task indexes somatic awareness only.

It is of particular interest that ADHD inattentive symptoms directly predicted binge/disinhibited eating, independent of any influence on mood and awareness and reliance on internal hunger/satiety cues, suggesting that these pathways are not the sole mechanisms through which inattentive symptoms are associated with binge/disinhibited eating pathology. Several experimental studies have shown that distraction while eating (e.g. watching television or playing a computer game) affects the memory encoding of a meal and is associated with increased later snack intake (Higgs & Woodward, 2009; Mittal, Stevenson, Oaten, & Miller, 2011; Oldham-Cooper, Hardman, Nicoll, Rogers, & Brunstrom, 2011). Conversely, attentive eating has been found to enhance memory encoding and reduce later snack intake (Higgs & Donohoe, 2011; Robinson, Kersbergen, & Higgs, 2014). It is therefore plausible that individuals with pronounced inattentive symptoms of ADHD may be easily distracted when eating, resulting in an impaired memory for recent eating, and subsequent overeating, especially in the presence of highly palatable foods. Further research is warranted to test this hypothesis.

In contrast to the findings from Study 1, the results from Study 2 provided evidence for a direct relationship between hyperactive/impulsive symptoms of ADHD and disordered eating, both binge/disinhibited eating and restrictive eating. These findings may be attributed to differences in sample population characteristics, and more specifically to the severity of ADHD symptoms. The Study 1 sample included some individuals who reported a previous diagnosis of ADHD by a healthcare professional (33.3% of the total sample). However, in Study 2, none of the participants reported a diagnosis of ADHD. Higher scores in the self-reported symptoms of ADHD assessed by the CAARS:S-SV suggest that participants in Study 1 had higher levels of ADHD symptoms than the participants in Study 2. It is therefore plausible in individuals with lower levels of hyperactivity/impulsivity symptoms of ADHD, there are other pathways that may link the symptoms to disordered eating in addition to negative mood. However, at higher levels of hyperactivity/impulsivity no direct relationship is observed, and the effect of hyperactive/impulsive symptoms of ADHD on disordered eating is entirely mediated by negative mood. Interestingly, in Study 2 while impulsivity, as assessed by the BIS, directly predicted binge/disinhibited eating there was no evidence for a direct association with restrictive eating, and thus, by implication, providing evidence for a specific contribution of impulsivity symptoms of ADHD to binge/disinhibited eating and hyperactivity symptoms of ADHD to restrictive eating. However, further studies are warranted to replicate these initial findings.

To the best of our knowledge, no studies have examined the influence of age, BMI and ADHD medication on the association between ADHD and disordered eating behaviour using formal moderation analysis. In addition, only 3 previous studies conducted a formal moderation to examine the effect of gender on the relationship between ADHD and disordered eating analysis (Bleck & DeBate, 2013; Davis, Cohen, Davids, & Rabindranath,

2015; Patte et al., 2016). Our findings are in agreement with the majority of previous evidence for a non-significant effect of gender on the association between ADHD symptomatology and binge behaviour (Kaisari et al. 2017). However, there is some limited evidence for a stronger association between the hyperactivity symptoms of ADHD and a drive for thinness in men than women (Grabarek & Cooper, 2008; Slane et al., 2010), which deserves further investigation.

Strengths and Limitations

Particular strengths of our studies are the relatively large sample sizes, the control for important confounds in the models and the simultaneous assessment of both binge/disinhibited eating and restrictive eating behaviour using well-validated measures both online and in a laboratory setting. The use of non-clinical samples ensures that confounds associated with clinical research (e.g. medication status) can be minimised, providing a practical approach that allows mechanism testing. Furthermore, recruitment from the general population enables generalisation of the findings to individuals in the general population who experience symptoms of ADHD and eating disorders but do not meet diagnostic criteria.

Despite these strengths, there are certain limitations of our studies. Firstly, the cross-sectional nature of the studies precludes causal inferences, and longitudinal studies would be better suited to investigate the temporal relationships between ADHD symptoms, negative mood, interoception deficits and disordered eating behaviour. In addition, despite the use of well-validated measures, most of the data collected in the present studies were based on self-reports, and it would therefore be valuable to validate these initial findings using objectively assessed measures. For example, the QbTest could be used to evaluate the core symptoms of ADHD (hyperactivity, inattention and impulsivity) (see <https://www.qbtech.com/>). In our studies, although we attempted to examine the specific contributions of hyperactive and

impulsive symptoms of ADHD to disordered eating, we are aware that conclusions should be tempered, as no objective measure of hyperactivity was used. Furthermore, while findings from our studies suggest that core symptoms of ADHD relate both to binge/disinhibited and restrictive eating pathology, it remains unclear whether binge/disinhibited eating and restrictive eating characterise two distinct groups within our sample population or whether the core symptoms of ADHD predispose individuals to increased eating pathology, with cycling between bingeing and restrictive eating. To the best of our knowledge, no study has examined longitudinally the complex interaction that may arise due to cyclical shifting between different subtypes of EDs (Fairburn & Harrison, 2003) and the change of predominant symptoms of ADHD that may occur throughout the lifetime (Holbrook et al., 2016). Our study is a valuable initial step towards elucidating the mediating factors that might explain the complex relationship between core symptoms of ADHD and disordered eating. Another potential issue for Study 1 is that internet-based data collection can result in selection or response bias. Low response rates can be a major concern that threatens the quality of web surveys (Couper, 2000; Crawford, Couper, & Lamias, 2001; Dommeyer & Moriarty, 1999), as in such cases the representativeness of the responders and validity of the results may be questioned. However, response rate was as high as 95.8% in the Study 1 online survey, suggesting that responders did not differ from non-responders. Furthermore, numerous studies have shown that questionnaire scores obtained online are comparable to those collected using traditional paper-and-pencil formats, and that psychometric properties of questionnaires are not adversely affected by computerised data collection (Mayr et al., 2012; Naus, Philipp, & Samsi, 2009). Indeed, it has been suggested that anonymous, web-based surveys may be more useful when collecting data on sensitive issues, such as eating- and body-related questions (Kays, Gathercoal, & Buhrow, 2012). Due to the self-administered nature of online surveys,

measurement error is a common problem in online data. The inclusion of trap questions and a self-report measure of study engagement in both studies allowed us to assess the quality of the collected responses, and take an informed decision about careless responses that could lead to biased results. Finally, despite the fact that the studies were advertised so that both males and females could take part, the sample in both studies comprised mainly female participants. There is also a potential sampling bias, as in both studies the participants were mainly highly educated adults. Therefore, future studies are warranted to replicate and extend the current findings in more representative and equally distributed samples of males and females.

Clinical Implications

The present findings suggest that prevention and intervention programs for eating pathology would be likely to benefit from treating mood disorders and interoception deficits in individuals who score highly on ADHD symptomatology. The direct relationship we identified between inattentive symptoms of ADHD and binge/disinhibited eating suggests that treatments and behavioral therapies that directly target attention deficits may be particularly effective in the management of both ADHD and binge eating disorder. Recently, lisdexamfetamine, a central nervous system stimulant used for the treatment of ADHD, has been approved for the treatment of moderate to severe binge eating (US Food and Drug Administration, 2015). It is unclear if this medication would be effective for individuals with ADHD who exhibit restrictive eating, and assessment of eating behaviours would be an important factor to consider when making treatment decisions. Ultimately, a better understanding of the range of specific eating problems experienced by individuals with ADHD and their underlying mechanisms will facilitate more effective and personalised treatment. Finally, as mood disorders are associated with the development of a range of psychopathologies including EDs (Casper, 1998; Fornari et al., 1992), interventions targeting

mood regulation may provide a transdiagnostic approach to resilience and mental health promotion programs, and the prevention and treatment of psychopathology more broadly.

Conclusion

In conclusion, the present studies showed that in two independent adult samples, ADHD symptoms were positively related to disordered eating, including both binge/disinhibited and restrictive eating and that negative mood mediated the relationships. Deficits in awareness and reliance on internal hunger/satiety signs provided another mechanistic pathway of association between inattentive symptoms of ADHD and disordered eating, especially binge/disinhibited eating. Notably, in both studies inattentive symptoms of ADHD were directly related binge/disinhibited eating. These findings could have important implications for prevention and early intervention programmes, which might usefully focus on mood regulation in individuals with ADHD symptoms at risk for developing disordered eating. Further investigation of the role of the inattentive symptoms of ADHD in disordered eating may be helpful in developing novel treatments for both ADHD and binge eating.

Chapter 5:

Investigation of eating behaviour and associated neural correlates in individuals with symptoms of Attention Deficit Hyperactivity Disorder (ADHD) compared to controls

5.1 Abstract

Evidence suggests an association between ADHD and disordered eating. However, experimental research in adults with ADHD is scarce, and most studies are based on questionnaire data. In this study, a between-subjects design was used to assess eating behaviour in individuals with high ADHD symptoms (31 ADHD⁺; 15 males, 16 females) compared to a matched control group (27 Controls; 13 males, 14 females). Eating behaviour was assessed using laboratory measures of food intake in conjunction with questionnaire measures. In addition, performance based measures (e.g. go/no go, attentional tasks) were used to assess the involvement of specific cognitive constructs (e.g. attention, inhibition) in eating behaviour. Neural correlates of eating behaviour were also investigated using fMRI. No main effect of group was found on laboratory measures of food intake. However, there was a trend for a significant interaction between gender and group on intake of a pasta lunch; ADHD⁺ females tended to consume less pasta than control females. Inattentive symptoms of ADHD were found to directly predict subjectively assessed binge/disinhibited eating behaviour. The results revealed for first time that inattentive symptoms of ADHD predicted overeating behaviour, assessed via consumption of a pasta meal in a laboratory setting, but this relationship was dependent upon gender. Thus, inattentive symptoms of ADHD predicted pasta intake only in males. Attention to food cues did not differ between controls and ADHD⁺ subjects. Although ADHD⁺ subjects reported higher levels of impulsivity on a self-report measure, analysis of responses to the go/no go task was suggestive of compensatory brain activation in ADHD⁺ adults to sustain normal response inhibition function. However, neural response to food cues, especially in the insula to high calorie food items, indicated potential reward-related differences between controls and ADHD⁺ individuals. Further, research is required to validate these initial findings.

5.2 Introduction

Attention Deficit Hyperactivity Disorder (ADHD) is one of the most debilitating childhood disorders, defined by age inappropriate impulsiveness, inattention, and hyperactivity, with symptoms of ADHD persisting into adulthood in about 75% of cases (Biederman, Petty, Clarke, Lomedico, & Faraone, 2011). Accumulating evidence suggests that there is a strong association between ADHD and eating disorders (EDs)/disordered eating (Kaisari, Dourish, & Higgs, 2017; Levin & Rawana, 2016; Nazar et al., 2016). However, whether ADHD and/or core symptoms of ADHD relate to specific subtypes of disordered eating remains uncertain. Overall, there is consistent evidence for an association between ADHD and overeating or binge eating behaviour, but the evidence on an association between ADHD and restrictive eating is more limited (for a review see Kaisari et al., 2007).

Impulsivity, is central to ADHD and has also been linked to overeating (Guerrieri, Nederkoorn, & Jansen, 2007; Guerrieri, Nederkoorn, Stankiewicz, et al., 2007), overweight/obesity (Braet, Claus, Verbeken, & Van Vlierberghe, 2007; Nederkoorn, Smulders, Havermans, Roefs, & Jansen, 2006; Ryden et al., 2003) and EDs, mainly of bingeing/purging subtype (*e.g. Bulimia Nervosa, Binge Eating Disorder*) (Waxman, 2009). Furthermore, impulsivity symptoms of ADHD have been consistently positively associated with bulimic and binge eating behaviour (Cortese et al., 2007; Mikami et al., 2010; Mikami, Hinshaw, Patterson, & Lee, 2008; Muller, Claes, Wilderjans, & de Zwaan, 2014), leading to the hypothesis that deficits in impulse control underpin the relationship between ADHD and EDs/disordered eating. However, impulsivity is a multifactorial concept that according to Dawe and Loxton (2004) involves at least two components: (a) reward sensitivity, the drive for appetitive or rewarding stimuli and (b) impaired inhibition, disinhibited behaviour without regard for the consequences. Indeed, a classical neuropsychological model of ADHD is the

dual pathway model that proposes two intertwined but separable neuropsychological pathways towards ADHD behaviour: (i) a failure of cognitive functioning and (ii) an altered motivational/reward related pathway (Sonuga-Barke, 2003). The dysfunctional cognitive functioning pathway is mainly characterized by deficits in inhibition and working memory, and the motivational pathway is characterized by an altered reward sensitivity (Sonuga-Barke, 2003). Taken together, these findings suggest that deficient inhibitory control (e.g. the ability to inhibit goal-irrelevant behaviours and cognitions) along with altered reward sensitivity may increase the risk of individuals with ADHD to develop disordered eating, especially overeating/binge eating behaviour. However, no studies to date have been conducted to specifically assess this dual pathway of disordered eating in ADHD.

Poor inhibitory control in ADHD has been reported in numerous studies (Alderson, Rapport, & Kofler, 2007; Aron & Poldrack, 2005; Bari & Robbins, 2013; Gilbert, Isaacs, Augusta, Macneil, & Mostofsky, 2011; Nigg, Butler, Huang-Pollock, & Henderson, 2002; Suskauer et al., 2008; Wodka et al., 2007). However, to the best of our knowledge only one study to date (Steadman & Knouse, 2016) has been conducted to investigate whether deficits in inhibitory control, as assessed via a go/no go task, mediated the relationship between ADHD symptoms and disordered eating in adults, namely binge eating behaviour. The results of this study were negative but the authors used non-disease specific stimuli in the go/no go task (e.g. letters). This may be significant as findings from the ED literature support greater impairments in inhibitory control in ED patients when confronted with disease-salient stimuli (e.g. food/eating) than general stimuli (Wu, Hartmann, Skunde, Herzog, & Friederich, 2013), suggesting that the use of context-specific stimuli may be more informative. However, no studies to date have used a food-based stimuli task to assess inhibitory control in the context of food cues in ADHD.

Recently, variants of the go/no-go task using food pictures have found faster reaction times on a modified go/no-go-'XY' attention task when food pictures were presented behind the "go" targets (Meule, Lutz, Vogeley, & Kubler, 2012). In addition, positive associations were found between rate and/or number of commission errors and aspects of overeating, especially emotional eating (Jasinska et al., 2012) and snack intake as assessed in a laboratory setting (Price, Lee, & Higgs, 2016), as well as positive associations with BMI values (Batterink, Yokum, & Stice, 2010). These findings are confounded, however, by the lack of a neutral comparison condition (Batterink et al., 2010; Jasinska et al., 2012) or homogeneity of food stimuli (only high-calorie food items) (Meule et al., 2012; Price et al., 2016). Without these comparisons, it is difficult to determine the extent to which inhibitory control deficits are driven by food and more specifically certain types of food (e.g. highly palatable) that are commonly associated with overeating behaviour. As a result, critical processes that are involved into the mechanisms of overeating, binge eating behaviour may be overlooked. To investigate the specific influence of appetite food cues on self-control, in the current study we use a food go/no go task that uses pictures of low- and high-calorie food stimuli along with a neutral-based stimuli task.

Abnormalities in reward processing have also been implicated in the neuropsychology of disordered eating and overweight/obesity (Garcia-Garcia et al., 2013; Stice, Spoor, Bohon, Veldhuizen, & Small, 2008; Weygandt, Schaefer, Schienle, & Haynes, 2012). In ADHD, although dysfunctions in reward pathways have been suggested to link the disorder with EDs/disordered eating and overweight/obesity (Bazar, Yun, Lee, Daniel, & Doux, 2006; Cortese et al., 2016), the evidence is limited (Cortese & Vincenzi, 2012). Food-cue responsiveness has been a common method to assess reward sensitivity, especially in the context of obesity, and attentional bias has been used as a behavioural measure to describe

high-responsiveness to food cues (Field et al., 2016). However, food can capture attention both in a “bottom-up” (Castellanos et al., 2009), as a consequence of a conditioning process, and a “top-down” manner via working memory (Higgs, Dolmans, Humphreys, & Rutters, 2015; Higgs, Rutters, Thomas, Naish, & Humphreys, 2012; Rutters, Kumar, Higgs, & Humphreys, 2015), and it is therefore important to investigate the specific mechanisms involved in attentional biases (see Chapter 2 for a detailed review of the literature). Furthermore, although behavioural techniques allow researchers to explore cognitive processing differences between individuals that may predispose certain people to disordered eating behaviours, the use of fMRI data enables an exploration of the neurocognitive origins of these potential differences in cognitive processing. Indeed, assessment of whole-brain activation patterns associated with the presentation of food pictures (e.g. salient and familiar cues previously associated with food reward) using fMRI is a common method of studying food reward processing, especially in the context of satiety and obesity (Fletcher et al., 2010).

For example, Stoeckel et al. (2008) found that pictures of high-calorie foods produced significantly greater activation in obese compared to lean individuals in a large number of reward-related regions, such as the medial and lateral orbitofrontal cortex, amygdala, nucleus accumbens/ventral striatum, medial prefrontal cortex, insula, anterior cingulate cortex, ventral pallidum, caudate, putamen, and hippocampus. To date there is consistent evidence of hyper-activation of reward system in obese individuals in response to food cues, especially to high-calorie/energy dense food cues, even when satiated (for a review see Pursey et al., 2014). To investigate responsiveness to food cues in the current study we used a behavioural measure that allows assessment of attentional biases to food cues, and captures both automatic (bottom-up) and top-down guidance of attention, as well as an fMRI paradigm (picture rating task) that assesses neural responses to food cues in comparison to non-food cues.

It is also plausible that individuals with ADHD develop EDs/disordered eating behaviour as a means to cope with negative affect caused by the outcomes of attention difficulties (e.g. poor work performance) and/or impulsive responses at the cost of planned goals. In Chapter 4, we tested these hypotheses, and across two independent samples we provided evidence for a mechanistic pathway of association between both symptom types of ADHD (*inattentive and hyperactive/impulsive*) and disordered eating behaviour (*including both binge/disinhibited and restrictive eating*) via negative mood. In this study, we used self-report measures to assess mood changes associated with anxiety, depression and stress in ADHD as used in Chapter 4, along with visual analogue scales (VAS) that measure subjective responses related to negative, and positive mood. This is important as it enables assessment of mood changes related both to longer periods (anxiety, depression, stress), as well as shorter periods (within the testing period), and to test for specific associations with disordered eating behaviour.

The findings from Chapter 4 also provided evidence for another mechanistic pathway of association between ADHD and disordered eating, via deficits in awareness and/or reliance on hunger/satiety signals. Notably, increased inattentive symptoms of ADHD were found to be negatively associated with a self-report measure of awareness and/or reliance on internal hunger/satiety signals to guide eating behaviour, and decreased awareness and/or reliance on internal hunger/satiety signals significantly predicted disordered eating behaviour. However, when we used a performance-based measure task (e.g. the heartbeat perception task) to assess interoceptive sensitivity, no association was found with either the inattentive symptoms of ADHD or disordered eating behaviour. The current study, aims to further validate these initial findings via assessment of awareness and/or reliance on internal hunger/satiety signals both via a self-report and a performance-based measure as used in Chapter 4.

Taken together, research to date suggests at least three potential pathways of association between ADHD and disordered eating: (i) diminished inhibitory control, in conjunction with altered reward processing, (ii) mood changes associated with processing of negative feelings (*e.g. anxiety, depression, stress*), and (iii) deficits in awareness and/or reliance on hunger/satiety signals to guide eating behaviour. However, this evidence base is largely reliant on theoretical evidence derived either from the ADHD and/or overweight/obesity, and ED literature. Few studies to date have used an experimental design to investigate eating behaviour in ADHD using laboratory measures, and these were limited to children and/or adolescents (Hartmann, Rief, & Hilbert, 2013; Munsch, Hasenboehler, & Meyer, 2011; Wilhelm et al., 2011). Experimental research in adults with ADHD is scarce, and it is largely based on questionnaire data (Davis, Levitan, Smith, Tweed, & Curtis, 2006; Patte et al., 2016; Steadman & Knouse, 2016; Strimas et al., 2008), providing limited insight on the involvement of specific cognitive constructs (*e.g. inhibition*) and neural correlates of eating behaviour. Furthermore, most of the studies to date have primarily focused on females, and if males were included they generally comprised a small proportion of the total sample, thereby not allowing any tests of whether the effect of ADHD on disordered eating is dependent upon gender.

We addressed the methodological limitations of previous research by including: (1) laboratory measures of eating behaviour, in conjunction with questionnaire based measures of disordered eating, (2) performance based measures to assess inhibitory control (go/no go task) and reward processing (picture rating task, attentional tasks) in the context of food and non-food cues, as well as interoceptive sensitivity (the heartbeat perception task), (3) fMRI to examine potential difference in neural activity to food cues that could underpin differences in eating, and (4) equal numbers of male and female participants. To avoid confounds of clinical

research (e.g. medication) we recruited healthy participants, differing in presentation of ADHD symptoms. Two groups of participants were recruited based on findings from Chapter 4: (i) individuals with low ADHD symptoms, who comprised the control group, and (ii) individuals with high ADHD symptoms, who comprised the high ADHD symptoms group, ADHD⁺.

To investigate eating behaviour, a validated Universal Eating Monitor (UEM), was used (Yeomans, 2000). The UEM consists of a set of hidden scales connected to a computer that measures the weight of the plate at regular intervals as the participant consumes their meal. To match normal eating outside the laboratory, participants were asked to consume a lunch (*pasta meal*) from the UEM around their usual lunch time (13:00 -13:30 pm) until satiated. Following the procedure described by Thomas, Dourish, and Higgs (2015), to examine eating in the absence of hunger, twenty minutes after finishing their lunch participants were offered a snack food (*chocolate chip cookies*) from the UEM. As behavioural correlates of binge eating may include eating in the absence of hunger (Marcus & Kalarchian, 2003), especially of highly palatable food items, this experimental procedure, enabled assessment of eating behaviour both in the context of a typical meal (*lunch meal*), but also in the absence of hunger (snack intake), providing a potential behavioural measure of binge eating behaviour.

We hypothesised that the ADHD⁺ group will show more disordered eating behavior compared to controls as assessed via subjective self-report measures, and especially of the binge eating type. We also hypothesised that food intake, as assessed via the UEM would differ between the ADHD⁺ group and the control group, with ADHD⁺ individuals showing higher food consumption, especially during eating in the absence of hunger (*snack intake*). In addition, we predicted that the ADHD⁺ group compared to the control group would report

higher levels of negative mood and less accuracy in detection of internal bodily sensations, as measured by the heartbeat perception task. Awareness and/or reliance on internal/hunger satiety signals were also expected to differ between groups, with the ADHD⁺ group reporting lower scores.

At the behavioural level, we hypothesised that the ADHD⁺ group compared to the control group would show impaired inhibitory control, as assessed via the go/no go task, both during the neutral-based and the food-based task, but that the effect would be stronger in the context of food stimuli, especially for the high calorie food cues. We also hypothesised, that the ADHD⁺ group compared to the control group would show greater attentional bias to food cues, as measured by the attentional task. At the neural level, we hypothesized that the most substantial differences between groups would be observed in regions involved in inhibitory control and reward value coding. In particular, the ADHD⁺ group compared to the control group would show reduced activation in areas typically involved in inhibitory control during a go/no-go task, such as the right inferior frontal junction reaching into insula, supplementary motor area or anterior cingulate cortex, left caudate head, and right thalamus (for a review see Hart, Radua, Nakao, Mataix-Cols, & Rubia, 2013). Finally, we hypothesised that in response to food cues, especially high-calorie energy dense food cues, the ADHD⁺ group compared to the control group would show increased activation in areas typically involved in reward processing, such as the ventral striatum (particularly nucleus accumbens), the orbitofrontal cortex and the ventro-medial prefrontal cortex (Pursey et al., 2014). Although, we planned to investigate activation in reward-related areas using the picture rating task, it was hypothesised that during the go/no-go task, food stimuli versus non-food stimuli and high-calorie versus low-calorie food stimuli would also elicit increased activation in reward-related regions, especially in the ADHD⁺ group.

5.3 Materials and Method

Sample Size

To calculate the sample size of the current study, a power analysis was performed for the hypothesis of interest that the ADHD⁺ group will show abnormal brain activation patterns in regions relevant to response inhibition. Based on findings from Mulligan et al. (2011) there will be 90% power to detect a difference in brain activation in regions relevant to response inhibition (fronto-parietal networks) (Cohen's $d = 0.85$; with ADHD adults showing reduced activation compared to controls), using a one-tailed t-test (2 independent means) at the 5% significance level if we recruit 50 participants in total. Therefore, we aimed to recruit 25 individuals scoring high in ADHD symptomatology and 25 gender- and age-matched control individuals. Based on previous analogous studies, this sample size is also considered adequate to detect differences in neural activation patterns related to reward coding in response to food cues (Chechacz et al., 2009; Martens et al., 2013; Rothenmund et al., 2007; Stoeckel et al., 2009; Stoeckel et al., 2008).

Design

A between-subjects design was used. The cut-off values for the low and the high ADHD symptoms groups were based upon previous research. Specifically, based on the findings from Study 1 (of Chapter 4), individuals who scored on the ADHD Total Symptoms subscale of the CAARS Scale equal or more than 22 reported significantly more disordered eating behaviours than individuals who scored less than 22. Therefore, participants who scored equal or above 22 on the ADHD Total Symptoms subscale (score ranges from 0-54) of the CAARS Scale comprised the high ADHD symptoms group (ADHD⁺). To distinguish participants with high and low ADHD symptoms, individuals who scored very low on the ADHD Total Symptoms subscale of the CAARS Scale comprised the control, non-ADHD group. Results

from Study 1 (of Chapter 4) revealed that individuals who scored less than 12 on the ADHD Total Symptoms subscale of the CAARS Scale belonged to the lowest quartile. Therefore, participants who scored less than 12 on the ADHD Total Symptoms subscale of the CAARS Scale comprised the control, non-ADHD group.

Participants

Students from the University of Birmingham, UK were recruited in exchange for course credits or cash payment through posters, posts on social media channels and adverts on the University Research Participation Scheme. Individuals were invited to take part in an fMRI study examining the neural correlates of measures of eating behaviour in adults with symptoms of ADHD compared to control individuals. To be able to take part in the study participants were required to be fluent in English and meet all of the following screening criteria: scoring within the specified cut-off values on the ADHD Total Symptoms subscale of the CAARS Scale, right-handed, non-smokers, non-diabetic, no food allergies, not vegetarian, women not pregnant or breastfeeding, stable body weight ($\pm 5\%$) for at least 3 months prior to taking part in the study, not severely obese (BMI range: 18.50 - 34.90 kg/m²), medication-free during the time of the study, no current clinical depression and no contradictions to fMRI scanning (e.g. the presence of a pacemaker). From September 2016 to March 2017, a total of 490 individuals expressed an interest in the study. Fifty-eight individuals met all the required criteria and comprised the final sample of the study; 31 ADHD⁺ and 27 controls. All participants provided informed consent before participation. The procedures used in this study were approved by the University of Birmingham Research Ethics Committee.

Measures

Interoceptive sensitivity: the heartbeat perception task. The heartbeat perception task has been described in detail in Chapter 4. In brief, interoceptive sensitivity was measured

using a heartbeat perception score, calculated as the mean score across two heartbeat perception intervals (45s, 60s) according to the following transformation: $\frac{1}{2} \sum (1 - (|\text{recorded heartbeats} - \text{counted heartbeats}|) / \text{recorded heartbeats})$. The heartbeat perception score varies between 0 and 1. The maximum score of 1 indicates absolute accuracy of heartbeat perception.

fMRI tasks. Before the imaging session, participants were familiarized with the fMRI paradigms through practice on a desktop computer. The order of the paradigms was fixed across participants. Thus, participants were first asked to complete the picture rating paradigm followed by the go/no-go paradigm. Results of the picture-rating paradigm have not yet been analysed. However, a brief description of the task is provided below to provide a clear presentation of the experimental procedure.

The picture rating paradigm. Participants were asked to look at pictures of food and visually matched (in terms of shape, complexity, brightness and colour) control pictures during fMRI scanning. The stimuli were adapted from a previous study looking at passive responses to food pictures in diabetic patients (Chechacz et al., 2009). In the present study, the task was updated and while each image was on display to subjects in the scanner, they were asked to immediately rate how ‘appealing’ each picture was to them on a scale of 1–5 using a hand-held keypad (1 = not at all, 2 = not really, 3 = neutral, 4 = a little, 5 = a lot). The appeal rating was thus made and recorded simultaneously with the stimulus presentation used for fMRI activation. The task was completed in 3 blocks, each one lasting approximately 7 minutes.

The go/no-go paradigm was designed to examine inhibition of prepotent responses (response inhibition). The task is described in full elsewhere in Chapter 4. Briefly, participants were asked to complete two tasks: (a) a neutral go/no-go task and (b) a food

go/no go task. In the neutral task, sports equipment and toiletries images were used; there were two conditions, with the sports equipment images comprising the “go” stimuli in one condition (toiletries images: “no go” stimuli) and the “no go” stimuli in the other condition (toiletries images: “go” stimuli). In the food-based task, high-calorie food and low-calorie food images were used. Similarly, to the neutral task there were two conditions, with the high-calorie food images comprising the “go” stimuli in one condition (low-calorie food images: “no go” stimuli) and the “no go” stimuli in the other condition (low-calorie food images: “go” stimuli). Examples of high-calorie food items included pictures of hamburger, pizza, chocolate cake, ice cream. Examples of low-calorie food images included broccoli, celery, kiwi fruit and raspberries. Participants were informed that the low-calorie food items were always pictures of fruits and vegetables. Two functional runs were carried out. Each run consisted of 160 trials (in two blocks of 80; 20 trials/condition) for the neutral task and the same for the food task, and lasted approximately 7 minutes. Images were presented using a ratio of 80% “go” to 20% “no-go” trials to create a prepotent “go” response. Each trial was presented for 750 ms and was preceded by a fixation cross for 1000 ms. A rest period of 8000ms followed each condition. Subjects were instructed to respond with a button press to explicitly instructed “go” stimuli and withhold any response to “no-go” stimuli, and to respond as quickly and accurately as possible. Reaction times were measured from the beginning of trial onset and collected with a fiber-optic response box system. The “go” and “no-go” categories were presented in a randomised order. Presentation order of the food and neutral tasks was randomised. Stimuli were presented visually using the E-Prime 2.0 software (Psychology Software tools, Inc., Sharpsburg, PA, USA; see Figure 5.1) and were displayed using a video projector that illuminated a rear projection screen located at the end of the

magnet. Subjects viewed stimuli through an adjustable mirror attached to the head coil. MRI acquisition was synchronised with the paradigm.

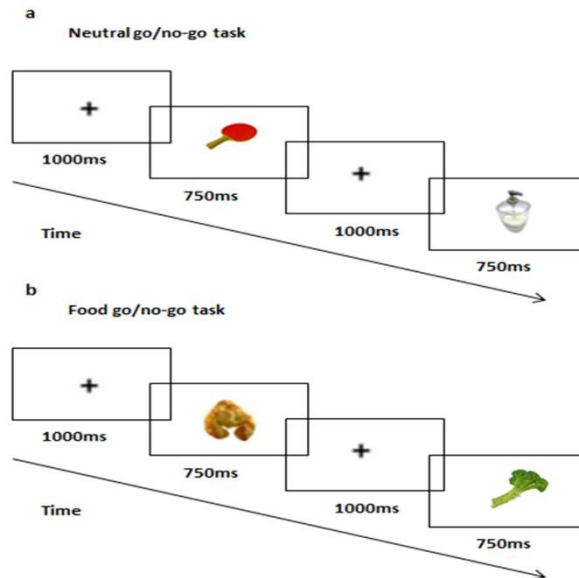


Figure 1. Go/no-go task: A total of 320 trials were presented, 160 (in two blocks of 80; 20 trials/condition) for the neutral task (a) and the same for the food task (b). Images were presented using a ratio of 80% ‘go’ to 20% ‘no-go’ trials to create a prepotent ‘go’ response. Each trial was presented for 750 ms and was preceded by a fixation point for 1000 ms. The go and no-go categories were presented in a randomised order. Presentation order of the food and neutral tasks was randomised.

Attentional tasks. The tasks are described in full in Chapter 2.

Universal Eating Monitor (UEM). In order to examine the microstructure of eating behaviour (e.g. eating rate), a Sussex Ingestion Pattern Monitor (SIPM), a validated Universal Eating Monitor (UEM) (Yeomans, 2000) was used. The setup consists of a balance (Sartorius Model CP4201; Sartorius Ltd, Epsom, UK; 0.1 g accuracy) placed under a table, projecting through the surface. A placemat on the table was placed on the top of the balance to create a more naturalistic eating environment. The balance was connected to a laptop computer. Using the procedure described by Thomas et al. (2015), the test meal consisted initially of an ad libitum lunch followed by an ad libitum snack intake to investigate eating in the absence of

hunger. To ensure satiation after the consumption of the lunch meal, participants were asked to take a break of 20 min before the snack food was offered (Castellanos et al., 2009; Nijs, Muris, Euser, & Franken, 2010). As microstructural intake data can be lost from the UEM due to participants unknowingly leaning on, or lifting the plate of food off the balance, before the offer of the lunch meal and the snack food, participants were made aware of the use of the UEM (Thomas et al., 2015). Participants were informed that the main aim of this task was to assess changes in hunger, fullness and pleasantness ratings within a meal. Based on previous research, awareness of the UEM is not expected to have a significant impact on food consumption (Thomas et al., 2015; Westerterp-Plantenga et al., 1990; Westerterp-Plantenga, Wouters, & Ten Hoor, 1991).

Ad libitum lunch. Plates containing 220g of pasta were placed on the mat and the amount of food eaten was recorded every 2 seconds. The UEM software (SIPM Software version 2.0.8) was configured to interrupt participants each time they had eaten 50g of the meal and ask them to complete VAS ratings of hunger, fullness and pleasantness of the pasta. After consumption of 150g pasta, participants were interrupted and the plate was replaced with a fresh 220g plate of pasta. Participants were instructed to eat as much as they wished of the pasta meal. The test meal consisted of pasta shells in a tomato and herb sauce, both Sainsbury's U.K. own brand served at 55-60°C (233 kcal/200g serving).

Ad libitum snack intake. Bowls containing 80g of Maryland chocolate chip cookies (390 calories/ 80g serving; purchased from Sainsbury's UK) were placed on the mat and intake was recorded every 2 seconds. Each cookie was broken into 6-7 pieces to reduce the likelihood that participants could track the number of cookies they ate, an approach used in previous research (Higgs & Woodward, 2009). The amount of cookies served ensured that participants were provided with more of the snack than they were likely to consume (Higgs &

Woodward, 2009). The UEM software (SIPM Software version 2.0.8) was configured to interrupt participants each time they had eaten 10g of cookies and ask them to complete VAS ratings as described earlier for the pasta meal. After consumption of 60g cookies, participants were interrupted and the bowl was replaced with a new one containing 80g of cookies. Participants were instructed to eat as much as they wished of the snack food.

Self-Reported Measures

Questionnaire measures for disordered eating, awareness and reliance on internal hunger/satiety cues, negative mood, alcohol and drug use were the same as described in Chapter 4. The Cronbach's alpha values in the present study are summarised in Table 1.

Impulsivity. *The Barratt Impulsivity Scale (BIS)* (Patton & Stanford, 1995), a 30-item self-report questionnaire, was used to assess impulsivity. The questionnaire has been described in full in Chapter 4. In the present study, the Cronbach's alpha was 0.88 for the total score.

Table 1*Cronbach's Alpha Values for the Questionnaire Measures*

Measures	Cronbach's alpha
<i>Disordered Eating</i>	
DEBQ - Emotional eating	0.96
DEBQ - External eating	0.84
DEBQ - Dietary Restraint	0.93
LOCES	0.89
BES	0.86
BITE	0.88
EAT Total score	0.81
EAT - Dieting	0.81
EAT – Bulimia ^{\$}	0.61
EAT - Oral Control [!]	0.09
<i>Awareness and reliance on internal hunger/satiety cues</i>	
RIH	0.78
<i>Negative mood</i>	
HADS - Anxiety	0.85
HADS - Depression	0.74
PSS	0.84
<i>Covariates</i>	
SMAST [£]	0.67
DAST-10 [^]	0.34

^{\$} Based on total 5 items. One item had zero variance and was removed. [!] Among the 7 items, 3 items had almost zero variance, and the remaining 4 items had very low variance. [£] Based on total 7 scale items. Items 6, 7, 8, 10, 12 and 13 had zero variance and were removed. [^] Based on total 7 items. Items 7, 9, and 10 zero variance and were removed.

Procedure

The experimental procedure is summarised in Figure 2. Participants were asked to consume their regular breakfast between 1-2 hours before coming to the Research Unit, but to refrain from eating or drinking (including caffeinated beverages) anything else in the meantime for standardisation purposes. All participants were asked to arrive at the Research Unit at 10:00 am and the imaging session started at 11:00 am. This allowed for a 2-3 hours deprivation period, before the imaging session. We selected this deprivation period to capture an inter-meal state of hunger, and investigate individual differences in response to food during a time that most individuals approach their lunch meal time.

Screening. Those who expressed an initial interest in the study were guided to a website to complete a brief online screening questionnaire to determine if they were eligible for the study. Respondents were asked to leave their contact details (email address and telephone number) for a member of the research team to contact them. Only participants who met all the study entry criteria could take part in the study. All participants who completed the screening survey were sent an email to confirm the receipt of their responses and to be informed whether they were eligible for the study. In this email, a copy of the participant information sheet (PIS) and an informed consent form for the study were attached to allow eligible participants to make an informed decision about their participation in the study. If participants, after reading the PIS and the informed consent form, were still interested in taking part in the study an appointment was arranged in order for the study to take place.

Contact details of the Principal Investigator were available in the PIS and potential participants were informed that they could contact the researcher via email or telephone if they wished to discuss the study and ask any questions they might have had about the study. In the online questionnaire participants were asked to complete: (a) a basic demographics and

lifestyle questionnaire (e.g. current body weight, height, medication use) (b) an fMRI safety form (c) the Beck's Depression Inventory (Beck, Ward, Mendelson, Mock, & ERBAUGH, 1961) to assess current levels of depression and (d) the Conners' Adult ADHD Rating Scale-Self Report Screening Version (CAARS) (Conners, Erhardt, & Sparrow, 1999) to assess ADHD symptoms. Based on the CAARS score, participants were allocated to the appropriate study group. Online consent was obtained prior to completion of the online screening questionnaire.

Test day. A texting service (or call/email) was used to remind participants of their study visit appointments and the study requirements relevant to food consumption before the imaging session. Participants were informed that lunch would be offered to them after completion of the fMRI scanning. To check for compliance, upon arrival at the Unit, participants were asked to report their food intake until that time of the day using a dietary recall. Participants were also asked to complete baseline appetite (*hunger, fullness, desire to eat, thirst*) and mood (*happy, sad, energetic, tired, alert, drowsy*) measures on 100mm Visual Analogue Scales (VAS). Subsequently, the heartbeat perception task was completed and participants were familiarised with the fMRI paradigms through practice on a desktop computer. Before the imaging session participants were asked to complete another set of VAS scales. Once scanning was complete, participants were asked to leave the scanner and have a 10 minutes break before the completion of the computer-based attentional tasks. Participants were also asked to complete another set of VAS scales at this time point.

After the completion of the attentional tasks participants were asked to complete another set of VAS scales. Subsequently, participants were asked to move to another room where they were offered a lunch meal (pasta) from the UEM and instructed to eat as much as they wished. After consumption of the lunch, a 20 minutes break was allowed during which

participants could read a book or a magazine. After the break, participants were offered a snack food (chocolate chip cookies) from the UEM, and they were instructed again to eat as much as they wished. Before leaving participants were informed that the last part of the study included the completion of some questionnaires related to eating behaviour, psychological health and personality traits. To complete the questionnaires participants were left alone in a room. Questionnaires were computer-based using the online platform Qualtrics. Participants were informed that they could avoid answering any question with which they felt uncomfortable. In addition, they were informed that they could have short breaks between questionnaires if they wished. At the end of the test day, participants' height was measured, and before leaving participants were fully debriefed, thanked and compensated for their time spent participating in the study.

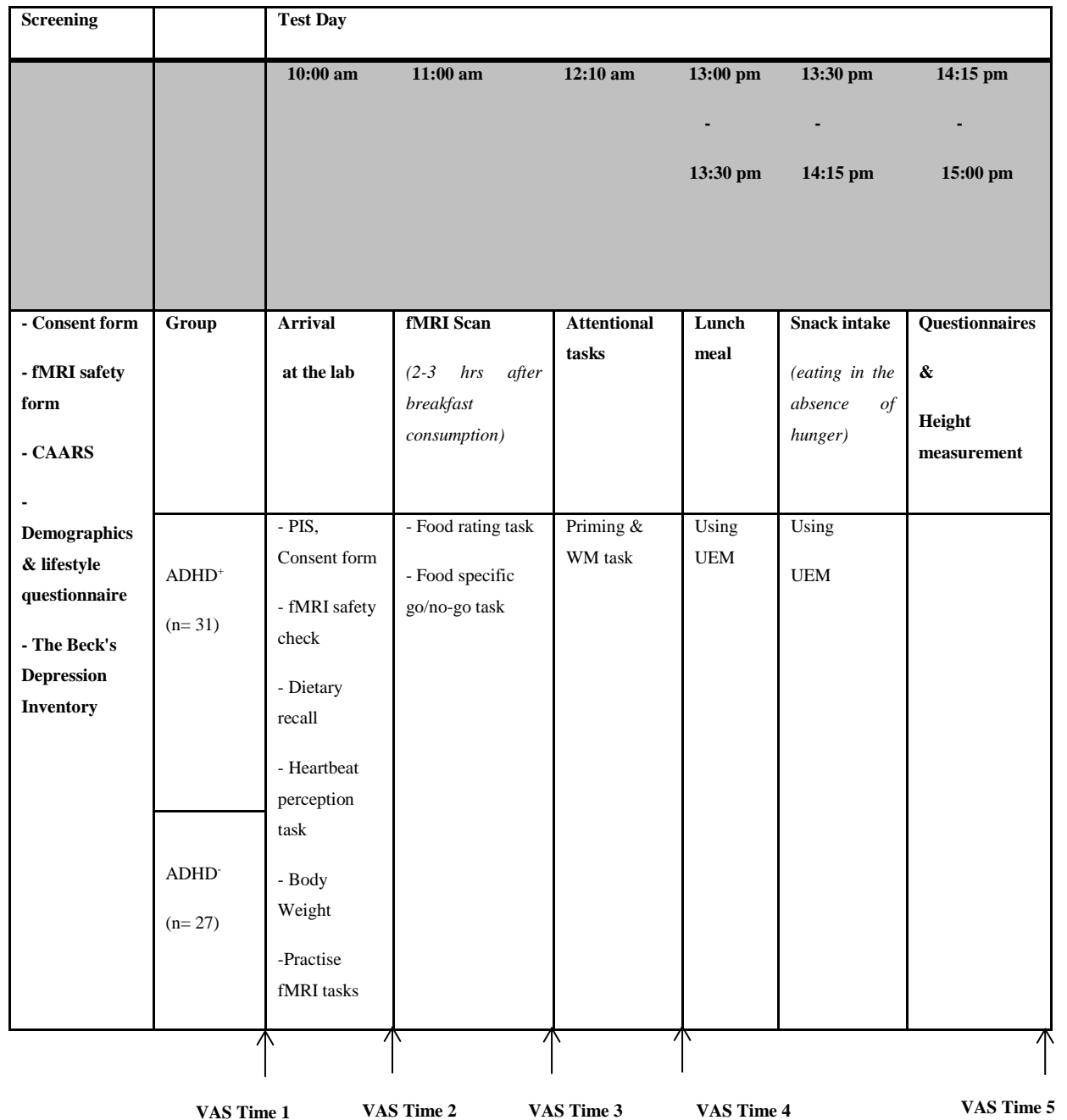


Figure 2. Experimental procedure.

fMRI data acquisition. Imaging data were acquired at the Birmingham University Imaging Centre (BUIC). A Philips Achieva 3.0T TX system with a standard 32-channel head coil was used to acquire T2-weighted echo-planar images (EPI) blood oxygenated level dependent (BOLD) contrast. Two functional scans were run. For each scan, forty-three transverse slices were acquired with 2 mm thickness, 1 mm gap between slices and a plane resolution of 3.0 mm \times 3.0 mm. We used a 2400 ms slice repetition time, 77° flip angle, and 30 ms echo time. The slices covered the most of the brain. A gradient echo field map (TE: short=9.2ms, long=11.5ms, flip angle=90°) with the same geometry as the EPI was acquired between the two functional scans. A high-resolution T1-weighted image was also acquired for each participant with 176 contiguous sagittal slices of 1 mm thickness and 7° flip angle. Time of repetition was 8.2 s and time of echo was 3.5 ms. The acquisition matrix was 256 mm \times 256 mm with voxel size of 1 mm \times 1 mm \times 1 mm.

fMRI data processing and statistical analysis. fMRI data were pre-processed and analysed using SPM12 (Wellcome Department of Cognitive Neurology, London, UK; www.fil.ion.ucl.ac.uk/spm). The field map toolbox in SPM was used to create a voxel displacement map for each participant, which was used in the unwarping procedure. The functional images were spatially realigned and unwrapped to correct for interactions between movement artifacts and field inhomogeneities. Images were also realigned in time to the middle slice. The functional images were then co-registered with structural image. Using the unified-segmentation algorithm the T1 structural image was segmented into different tissue types, the outputs was then used in the DRATEL toolbox, to create a group template, which was normalized to MNI space. Finally, the normalised images were smoothed with a Gaussian kernel of 8 mm.

A whole-brain voxel-based analysis was performed with SPM12. We used a general linear model to estimate the response for each of the 8 conditions in each session: go for HC food items, go for LC food items, go for sport items, go for toiletry items, no-go for HC food items, no-go for LC food items, no-go for sport items and no-go for toiletry items. The experimental design matrix included a regressor depicting the stimulus' onset in each trial of each condition. To account for the delay in the hemodynamic function, the regressors were convolved with the canonical haemodynamic response function. Movement parameters extracted from the realignment procedure and harmonics that capture low-frequency changes in the signal (i.e. high pass filtering with frequency cut-off of 1/128 Hz), typically associated with physiological and mechanical noise were also included in each subject's model.

For each participant, we computed the average response per condition across the two sessions. These data were used in the second level analysis, where subjects were treated as random factors. The design was a mixed design with the eighth condition as a repeated measure and the group as a between measure. In the analysis, we used main effects to identify regions of interest, the effects of group were explored in more detail within these regions using SPSS. ANOVA and t-tests were used for data that were normally distributed and the Mann Whitney U test was used, when the data were not normally distributed. The contrasts used to identify the regions of interests were: main effect of trial (go>no-go; no-go>go), main effect of stimuli type (food > non-food; non-food>food) and the effect of calorie content (HC>LC; LC>HC). Results of each of these contrasts were corrected for multiple comparisons using cluster level correction (clusters > 40 voxels showing $p < 0.001$ uncorrected). Anatomical labelling was achieved using the Anatomical Automatic Labelling toolbox (Tzourio-Mazoyer et al., 2002).

To assess group related differences in the identified regions, we extracted the beta estimates for each condition per participant, from a 3mm sphere around the peak activation of the group. These individual estimated effect sizes (beta values) were entered into a 2 (go and no-go) x 4 (HC, LC, sports, toiletry) repeated measures ANOVA with group (ADHD⁺ vs. control) as a between-subjects factor. To avoid double dipping the analysis focused on the effects that include the group factor; which are orthogonal to the way the regions were identified.

Analysis Plan for Questionnaire and Behavioural Data

Data reduction.

Disordered eating. As in Chapter 4, composite scores were created for ‘Binge/Disinhibited Eating’ and ‘Restrictive Eating’.

Negative mood. A composite score was calculated as in Chapter 4. In the current study, the extracted component accounted for 82% of the variance in the three emotional scales, and all three scales loaded strongly on this factor (range from 0.902 to 0.919).

VAS mood ratings. Mood ratings related to negative states of mood (*sad, tired, and drowsy*) were averaged into a composite measure for the analysis. Similarly, a composite measure was created for mood ratings related to positive states of mood (*happy, energetic, and alert*).

Data were analysed using SPSS Statistics 22 (IBM). Independent t-tests were used to compare continuous demographic variables (e.g. age, BMI), and ADHD symptomatology between control and ADHD⁺ groups. In case of unequal variances, as indicated by Levene’s test, adjusted degrees of freedom were reported. The χ^2 -test of independence was used to compare group gender and racial composition using defined categories of White, Black, Asian or Mixed/multiple ethnic groups. Appetite and mood VAS ratings were analysed using

repeated measures ANOVA with group as a between-subjects factor, and gender and BMI as covariates. Repeated measures ANOVA with food type as a within-subjects factor, and BMI as a covariate was also used to examine the effect of gender and group on food intake. To analyse the attentional tasks a similar approach to that described in Chapter 2 was followed.

Performance measures on the go/no-go task were analysed independently using repeated measures ANOVA with stimuli as a within-subjects factor and group as a between-subjects factor. Bonferroni corrections were used for all post-hoc t-tests. Separate two-way ANOVAs, with BMI as a covariate were conducted that examined the effect of gender and group on binge/disinhibited eating, restrictive eating, awareness and reliance on internal hunger/satiety cues, interoceptive sensitivity, negative mood, alcohol and drug use, and impulsivity. Moderation effects (*if any*) of gender were examined using PROCESS for SPSS (Hayes, 2013). Specifically, Model 1 was tested. Moderation is found if any of the tested interactions (e.g. inattentive symptoms of ADHD x gender) is statistically significant ($p < 0.05$). Continuous variables were mean centred as recommended by Howell (2013).

5.4 Results

Participants

Subject characteristics are summarised in Table 2. In total, 58 participants completed the study; 31 ADHD⁺ and 27 controls. No statistically significant differences existed in any of the studied baseline characteristics between groups (see Table 2 for details). The mean age of the ADHD⁺ subjects was 23.7 (SE = 0.9) and of the controls 23.0 (SE = 0.8). Both groups were comprised almost equal numbers of males and females, and most study participants were of White/White British and Asian/Asian British ethnicity. The mean BMI was 23.4 (SE=0.59) for the ADHD⁺ group and 22.9 (SE=0.64) for the control group. Most participants in both groups had a BMI within the normal range (71% of the ADHD⁺, 77.8% of the controls). A

smaller number of participants were classified as overweight/obese in both groups (28.9% of the ADHD⁺, 18.5% of the controls). Only one participant in the control group was classified as underweight.

Table 2*Baseline Characteristics of Groups*

	ADHD⁺		Controls			
	(n=31)		(n=27)			
Demographics	Mean	SE	Mean	SE	t(56)	p
	(Min –Max)		(Min –Max)			
Age (years)	23.7	0.9	23.0	0.8	-0.6	0.6
	(18-38)		(18-33)			
Gender	n	%	n	%	x²(1)	p
Female	16	51.6	14	51.9	0.1	1.0
Male	15	48.4	13	48.1		
Ethnic Background	n	%	n	%	x²(4)	p
White/White British	16	51.6	12	44.4	6.0	0.2
Asian/Asian British	6	19.4	10	37.0		
Black/African/Caribbean /Black British	1	3.2	3	11.1		
Mixed/multiple ethnic groups	3	9.7	1	3.7		
Other ethnic group	5	16.1	1	3.7		
Weight	Mean	SE	Mean	SE	t(56)	p
	(Min –Max)		(Min –Max)			
BMI (kg/m ²)	23.4	0.59	22.9	0.64	-0.5	0.6
	(19.4-30.4)		(18.0-32.0)			
Weight category	n	%		%	x²(3)	p
Underweight	0	0	1	3.7	2.1	0.6
Normal weight	22	71.0	21	77.8		
Overweight	8	25.8	4	14.8		
Obese	1	3.2	1	3.7		

Current ADHD Symptoms

Table 3 shows the mean and SE, and minimum (min) and maximum (max) scores for the 4 subscales derived from the CAARS: S-SV by group. As expected, the ADHD⁺ group scored significantly higher on all 4 subscales.

Of the 58 individuals, only 1 (male) had received a diagnosis of ADHD (hyperactive/impulsive type; at the age of 4 years old), and reported no current and past year treatment with medication.

No gender differences were observed on average scores on the 4 subscales derived from the CAARS: S-SV in the control group (all $p>0.05$). However, in the ADHD⁺ group, females reported on average higher scores on the Inattentive Symptoms subscale than males ($M = 17.56$, $SE = 4.15$ vs. $M=13.40$, $SE = 4.44$; $t(29) = 2.7$, $p = 0.01$). No difference was observed on the Hyperactive-Impulsive Symptoms subscale ($t(29) = 0.11$, $p = 0.91$) and the ADHD Index subscale ($t(29) = 1.73$, $p = 0.10$). Females, in the ADHD⁺ group also reported on average higher scores on the Total ADHD Symptoms subscale than males ($M = 31.38$, $SE = 6.14$ vs. $M=27.07$, $SE = 5.08$; $t(29) = 2.12$, $p = 0.04$).

Table 3

Mean, SE, Min and Max Scores on the Four Subscales Derived from the CAARS: S-SV by group

Current ADHD Symptoms	ADHD⁺ (n=31)		Controls (n=27)			
	Mean (Min–Max)	SE	Mean (Min–Max)	SE	t(44)	p
Inattentive Symptoms (0-27)	15.5 (5-24)	0.9	3.5 (0-8)	0.4		<0.001
Hyperactive-Impulsive Symptoms (0-27)	13.8 (6-22)	0.6	3.1 (0-8)	0.4	t(47)	p <0.001
Total ADHD Symptoms (0-54)	29.3 (21-42)	1.1	6.6 (0-11)	0.6	t(46)	p <0.001
ADHD Index (0-36)	19.5 (12-32)	0.9	5.2 (0-16)	0.7	t(56)	p <0.001

Interoceptive Sensitivity: The Heartbeat Perception Task

The mean score of interoceptive sensitivity was 0.68 (SE = 0.04) for the control group and 0.69 (SE = 0.04) for the ADHD group.

A two-way ANOVA, with BMI as a covariate was conducted to examine the effect of gender and group on the heartbeat perception task. No main effect of group ($F(1, 53) = 0.04$, $p = 0.84$, $\eta^2 = 0.001$) or gender ($F(1, 53) = 1.17$, $p = 0.28$, $\eta^2 = 0.02$) was observed, nor was there a significant interaction between group and gender on heartbeat perception accuracy scores ($F(1, 53) = 0.03$, $p = 0.86$, $\eta^2 = 0.001$).

VAS Scales

Appetite. Appetite ratings (*hunger, fullness, desire to eat, thirst*) were analysed using repeated measures ANOVA with group as a between-subjects factor, and gender and BMI as covariates. As expected, for hunger ratings there was a main effect of time ($F(4,212) = 5.07$, $p = 0.001$, $\eta^2 = 0.09$), with subjects reporting increased levels of hunger approaching meal time (time 1 to time 4) and significantly decreased levels of hunger at the end of the testing day (time 5; after meal and snack consumption) (see Figure 3). There was also a trend for a significant effect of gender ($F(1,53) = 3.48$, $p = 0.07$, $\eta^2 = 0.06$), with males reporting on average greater hunger scores than females, but there was no main effect of group nor any interactions ($p_s > 0.05$). For fullness, there was a main effect of time ($F(4,212) = 2.84$, $p = 0.03$, $\eta^2 = 0.05$), with subjects' ratings for fullness showing an opposite pattern to that observed for hunger ratings with time (see Figure 3). No other main effects or interactions were found. Concerning Desire to eat, there was a main effect of time ($F(4,212) = 5.54$, $p = < 0.001$, $\eta^2 = 0.10$), with subjects' ratings following a similar pattern as that observed for hunger ratings, and a trend for significant interaction between group and time ($F(4,212) = 2.13$, $p = 0.08$) (see Figure 4). Desire to eat ratings increased approaching meal time (time 1 to time 4) for both the ADHD⁺ and control group, however there was a trend for the ADHD⁺ group at time 3 to score higher than the control group ($t(56) = 1.76$, $p = 0.08$). No other main effects or interactions were found. For thirstiness, only a marginal main effect of gender was observed ($F(1,53) = 4.06$, $p = 0.05$, $\eta^2 = 0.07$), with males reporting on average a higher score on thirstiness than females.

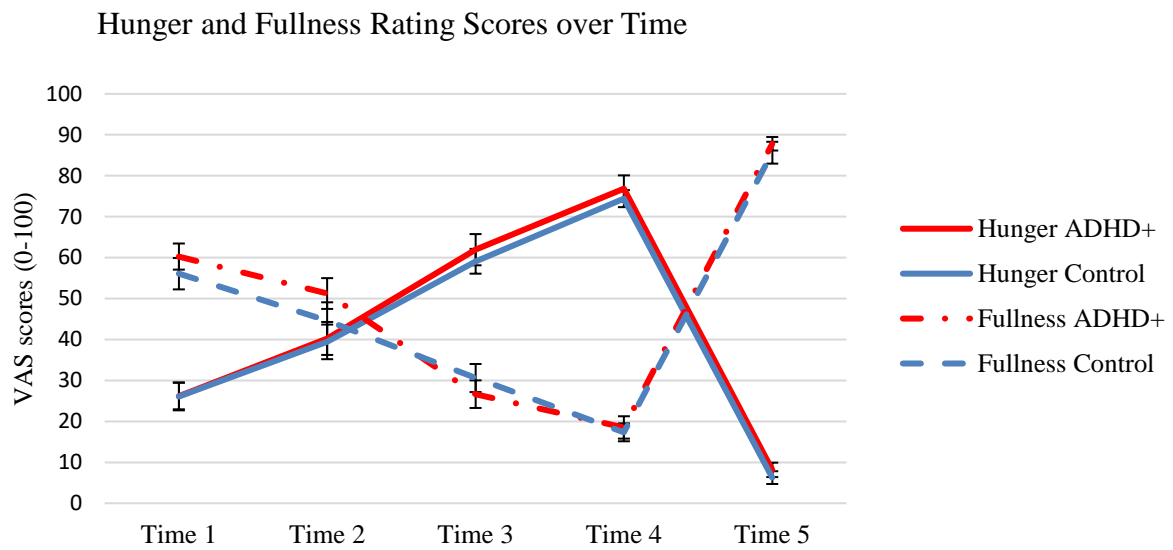


Figure 3. Hunger and fullness rating scores over time by group. Time 1: Baseline, Time 2: Before imaging session, Time 3: After imaging session, Time 4: Before meal, Time 5: End of testing day.

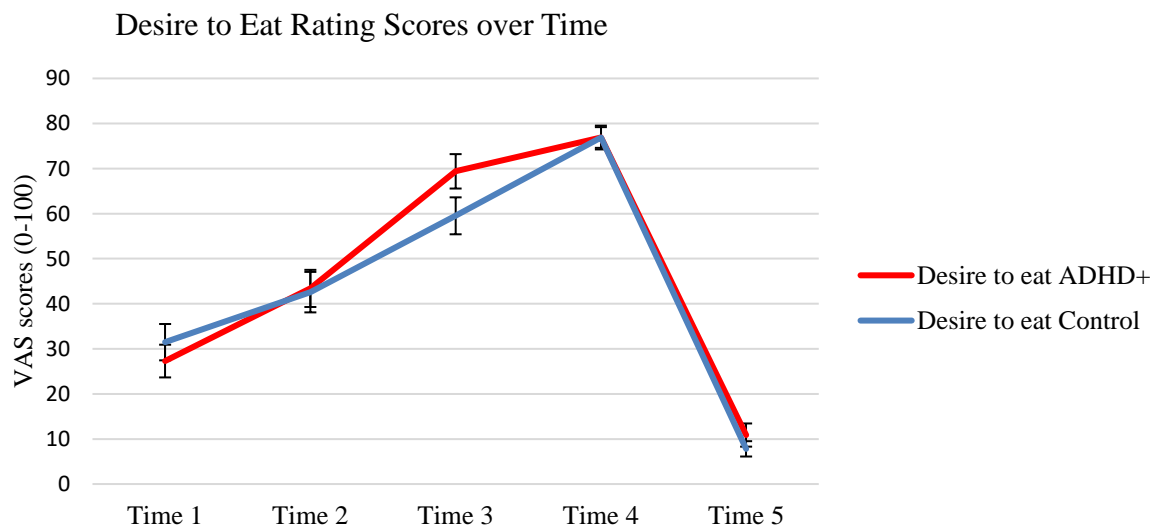


Figure 4. Desire to eat rating scores over time by group. Time 1: Baseline, Time 2: Before imaging session, Time 3: After imaging session, Time 4: Before meal, Time 5: End of testing day.

Mood. Mood ratings were analysed using repeated measures ANOVA with group as a between-subjects factor, and gender and BMI as covariates. Concerning mood ratings related

to positive states of mood (*happy, energetic, and alert*) no main effects of time and group were observed or interactions ($p>0.05$). There was a marginal main effect of gender ($F(1,51) = 3.89, p = 0.05, \eta^2 = 0.07$), with males on average reporting greater scores than females. However, concerning mood ratings related to negative states of mood (*sad, tired, and drowsy*), a significant main effect of group was observed ($F(1,51) = 4.69, p = 0.04, \eta^2 = 0.08$), with ADHD⁺ subjects scoring on average higher than controls. There was also a significant interaction between negative states of mood and ADHD⁺ status, ($F(4,204) = 2.43, p = 0.049, \eta^2 = 0.05$). As time was passing and approaching meal time (time 1 to time 4), the ADHD⁺ group scored significantly higher than the control group on all time points negative states of mood. However, at the end of the testing day (time 5; after meal and snack consumption), average negative mood scorings did not differ between groups (see Figure 5).

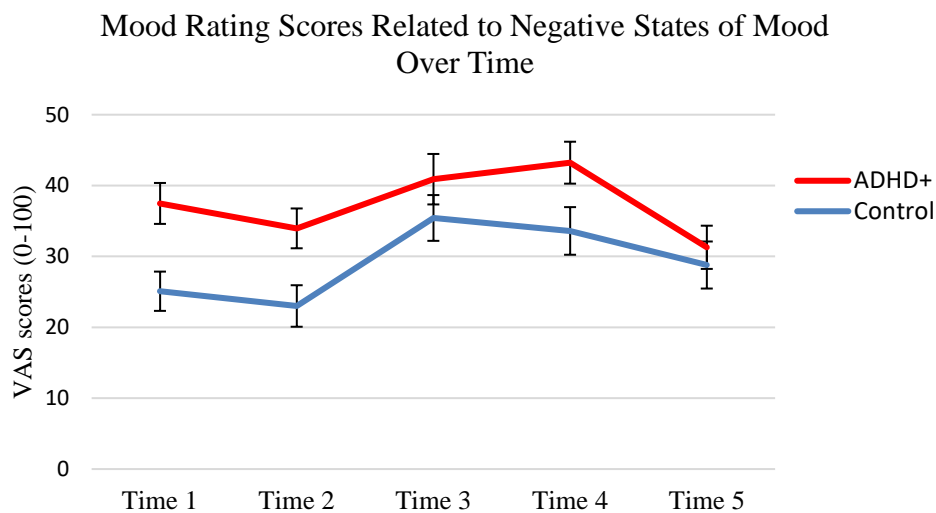


Figure 5. Mood rating scores related to negative states of mood over time by group. Time 1: Baseline, Time 2: Before imaging session, Time 3: After imaging session, Time 4: Before meal, Time 5: End of testing day.

Food intake. Repeated measures ANOVA with food type as a within-subjects factor, and BMI as a covariate examined the effect of gender and group on food intake. There was a significant interaction between food type, group and gender, $F(1,52) = 4.45$, $p = 0.04$, $\eta^2 = 0.08$. To break down this interaction, a separate two-way ANOVA, with BMI as a covariate was conducted to examine the effect of gender and group on pasta and cookie intake.

Pasta intake. No significant main effect of group was observed, $F(1,52) = 0.02$, $p = 0.89$, $\eta^2 = 0.00$. There was a significant interaction between gender and group on pasta intake, $F(1,52) = 4.37$, $p = 0.04$, $\eta^2 = 0.08$. Simple main effect analysis showed that pasta intake did not differ between control and ADHD⁺ males ($t(25) = -1.30$, $p = 0.21$). However, there was a trend for ADHD⁺ females to consume less pasta than control females ($t(28) = -1.90$, $p = 0.07$) (see Figure 6).

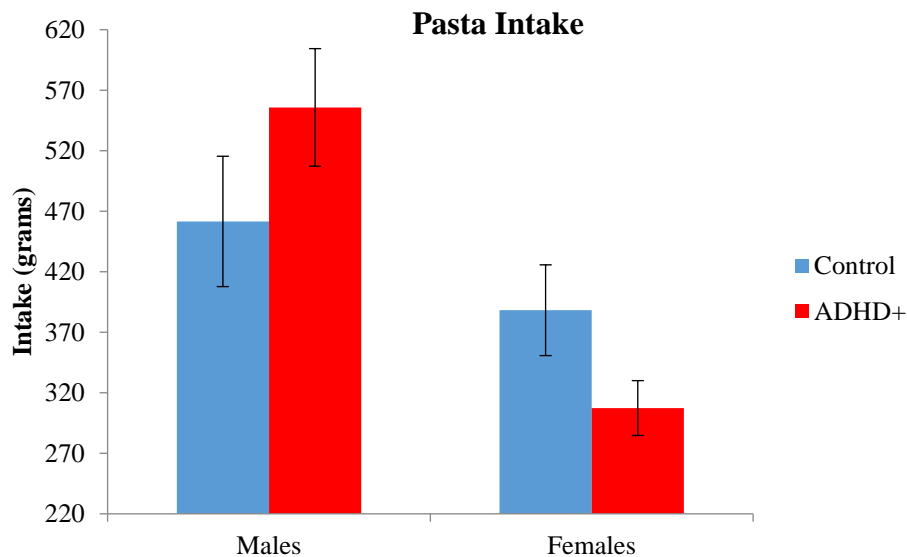


Figure 6. Mean pasta intake by group and gender.

Cookie intake. A significant main effect of gender was observed on cookie intake, $F(1,53) = 7.77$, $p = 0.01$, $\eta^2 = 0.13$, with males consuming on average significantly more cookies than females. However, no main effect of group, $F(1,53) = 1.21$, $p = 0.38$, $\eta^2 = 0.02$, or a significant interaction between gender and group was observed on cookie intake, $F(1,53) = 0.54$, $p = 0.47$, $\eta^2 = 0.01$ (see Figure 7).

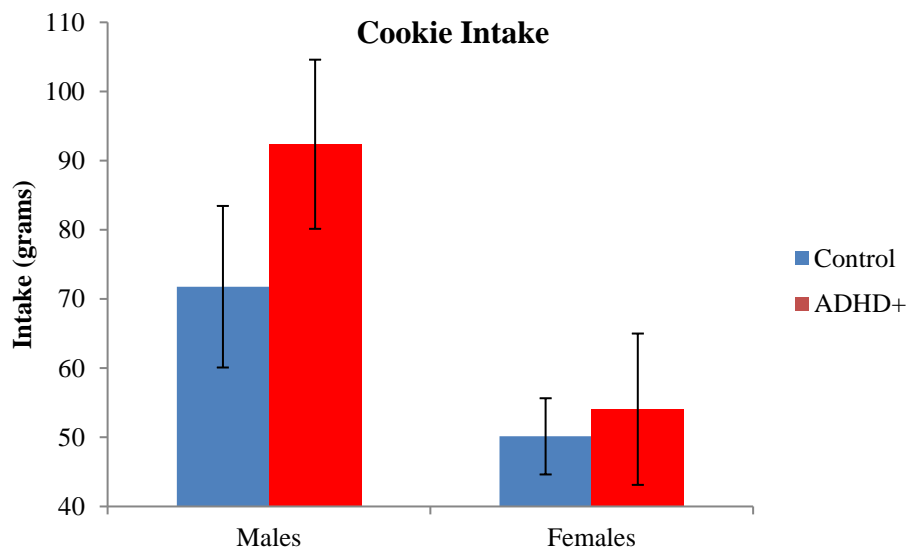


Figure 7. Mean cookie intake by group and gender.

Self-Reported Measures

Separate two-way ANOVAs, with BMI as a covariate were conducted that examined the effect of gender and group on binge/disinhibited eating, restrictive eating, awareness and reliance on internal hunger/satiety cues, negative mood, alcohol and drug use, and impulsivity.

Eating behaviour.

Binge/disinhibited eating. The mean score of binge/disinhibited eating was 17.09 (SE = 1.81) for the control group and 28.70 (SE = 2.77) for the ADHD group. A significant main

effect of group was observed, $F(1,53) = 11.69$, $p = 0.01$, $np2 = 0.18$, with ADHD⁺ subjects scoring significantly higher than control subjects ($t(51) = 3.51$, $p = 0.01$). A significant main effect of gender was also observed, $F(1,53) = 5.35$, $p = 0.03$, $np2 = 0.09$, with females scoring on average significantly higher than males ($t(51) = 2.11$, $p = 0.04$), but no interaction between group and gender was observed, $F(1,53) = 0.02$, $p = 0.90$, $np2 = 0.00$).

Restrictive eating. The mean score for restrictive eating was 6.26 (SE = 1.21) for the control group and 8.46 (SE = 1.21) for the ADHD group. A main effect of gender ($F(1,53) = 4.54$, $p = 0.04$, $np2 = 0.08$) was observed on restrictive eating, with females scoring on average significantly higher than males ($t(47) = 2.11$, $p = 0.04$), but no main effect of group or interaction between group and gender was observed ($ps > 0.05$).

Awareness and reliance on internal hunger/satiety cues.

The mean score for awareness and reliance on internal hunger/satiety cues was 3.96 (SE = 0.12) for the control group and 3.76 (SE = 0.12) for the ADHD group. No main effect of group or gender or interaction between group and gender was observed ($ps > 0.05$).

Negative mood.

The mean score for negative mood was 21.48 (SE = 2.08) for the control group and 31.29 (SE = 2.15) for the ADHD group. A main effect of group ($F(1,53) = 10.18$, $p = 0.002$, $np2 = 0.16$) was observed on negative mood, with ADHD⁺ subjects scoring significantly higher than control subjects ($t(56) = 3.26$, $p = 0.002$), but no main effect of gender or interaction between group and gender was observed ($ps > 0.05$).

Alcohol use.

The mean score for the SMAST was 0.74 (SE = 0.21) for the control group and 0.94 (SE = 0.26) for the ADHD group. No main effect of group or gender or any interaction between group and gender was observed ($ps > 0.05$).

Drug use.

The mean score for the DAST-10 was 0.74 (SE = 0.14) for the control group and 0.90 (SE = 0.20) for the ADHD group. No main effect of group or gender or any interaction between group and gender was observed ($p > 0.05$).

Impulsivity.

The mean of the BIS total score was 56.48 (SE = 1.70) for the control group and 72.03 (SE = 1.95) for the ADHD group. A main effect of group ($F(1, 53) = 34.64$, $p < 0.001$, $\eta^2 = 0.40$) was observed on impulsivity, with ADHD⁺ subjects scoring significantly higher than control subjects ($t(56) = 5.93$, $p < 0.001$), but no main effect of gender or any interaction between group and gender was observed ($p > 0.05$).

To investigate the effect of group (*if any*) on specific components of impulsivity, a repeated measures ANOVA with impulsivity (attentional, motor, non-planning) as a within-subjects factor, group and gender as a between-subjects factor and BMI as a covariate was conducted. There was a trend for a significant interaction between group and impulsivity, $F(2, 106) = 2.97$, $p = 0.06$, $\eta^2 = 0.05$; while the ADHD⁺ group scored higher on all subscales of the BIS (attentional, motor, no-planning) compared to the control group the effect was stronger for the attentional subscale ($t(56) = 7.72$, $p < 0.001$, $d = 2.03$; $t(56) = 3.58$, $p = 0.001$, $d = 1.05$; $t(56) = 3.73$, $p < 0.001$; $d = 0.99$, respectively).

Attentional bias.

Mean RTs (in milliseconds) to food and non-food cues for valid, invalid, and neutral trials, for both the priming and the WM tasks, are presented in Figure 8 for ADHD⁺ and control participants separately. RTs were slower in the WM than the priming task ($F(1, 56) = 33.19$; $p < 0.001$, $\eta^2 = 0.37$) suggesting that the participants were performing these tasks differently. There was a main effect of validity ($F(2, 112) = 154.93$; $p < 0.001$, $\eta^2 = 0.74$),

whereby RTs were faster for valid trials than the neutral and invalid trials, and faster for neutral compared to invalid trials (all $p < 0.01$). There was also a main effect of cue ($F(1, 56) = 36.33$; $p < 0.001$, $\eta^2 = 0.39$), whereby RTs for food cues were faster than RTs for non-food cues. There was a significant two-way interaction between task and cue ($F(1, 56) = 8.08$; $p = 0.006$, $\eta^2 = 0.13$), RTs were shorter for food cues in both the priming ($p = 0.01$) and WM task ($p < 0.001$), however the difference was smaller in the priming task. In addition, there was a significant two-way interaction between task and validity ($F(2, 112) = 35.34$; $p < 0.01$, $\eta^2 = 0.39$); RTs were shorter for valid trials compared to invalid trials ($p < 0.001$), and neutral trials ($p < 0.001$) in the WM task. A similar pattern was observed in the priming task (all $p_s < 0.001$), however, the difference between valid and neutral trials, and between valid and invalid trials was smaller. The two-way interaction between validity and cue ($F(2, 112) = 10.27$; $p < 0.01$, $\eta^2 = 0.16$) was also significant; RTs were shorter following food cues compared to non-food cues in the valid ($p < 0.001$) and neutral trials ($p < 0.001$), while no differences were observed in the invalid trials ($p > 0.05$).

To further explore the validity by cue interaction, RT for valid trials was subtracted from RT for invalid trials. Increased values on this score are suggestive of longer RT for invalid trials compared to valid trials, which translates to increased effort to disengage from the cued item when it was a distractor. Paired sample t-tests showed that when the cue was a food item the biasing effect was greater than when it was a non-food item for both the priming ($t(57) = 2.79$; $p = 0.0107$) and the WM task ($t(57) = 3.54$; $p = 0.001$). However, the biasing effect was significantly greater in the WM task than the priming task, both for the food cues ($t(57) = 4.83$; $p < 0.01$) and the non-food cues ($t(57) = 6.80$; $p < 0.01$), suggesting that holding a cue in WM has a significantly greater effect in biasing attention to visual similar stimuli even if it is irrelevant to the subsequent search task than just primed with a cue (e.g. by the

sight of the cue). Results from Chapter 2 revealed that overweight/obesity affects the degree to which holding food related information in WM biases attention to similar stimuli by affecting the ability to disengage from a cued item when it was a distractor. Therefore, in the present study BMI was entered as covariate to the model. Group and gender were also fixed factors. Results revealed no main effects or interactions for the biasing effect either for food or non-food cues, for both the priming and the WM task ($p > 0.05$).

The three-way interaction between task, validity, and cue ($F(2, 112) = 0.16$; $p = 0.86$, $\eta^2 = 0.003$) was not significant. The correction for gender did not change the three-way interaction ($F(2, 110) = 0.72$; $p = 0.49$, $\eta^2 = 0.01$). There was no significant effect of group ($F(1, 56) = 1.25$; $p = 0.27$, $\eta^2 = 0.02$), indicating that mean RTs from ADHD⁺ and control participants, were similar. There was no significant interaction between task and group ($F(1, 56) = 0.68$; $p = 0.41$, $\eta^2 = 0.01$), cue and group ($F(1, 56) = 0.62$; $p = 0.44$, $\eta^2 = 0.01$), validity and group ($F(2, 112) = 0.36$; $p = 0.70$, $\eta^2 = 0.006$), task, cue and group ($F(1, 56) = 0.07$; $p = 0.80$, $\eta^2 = 0.001$), task, validity and group ($F(2, 112) = 0.20$; $p = 0.82$, $\eta^2 = 0.004$), cue, validity and group ($F(2, 112) = 0.28$; $p = 0.75$, $\eta^2 = 0.005$). The four-way interaction between task, validity, cue and group was also not significant ($F(2, 112) = 1.34$; $p = 0.27$, $\eta^2 = 0.02$). Similarly, the correction for gender did not change the four-way interaction ($F(2, 110) = 1.33$; $p = 0.27$, $\eta^2 = 0.02$).

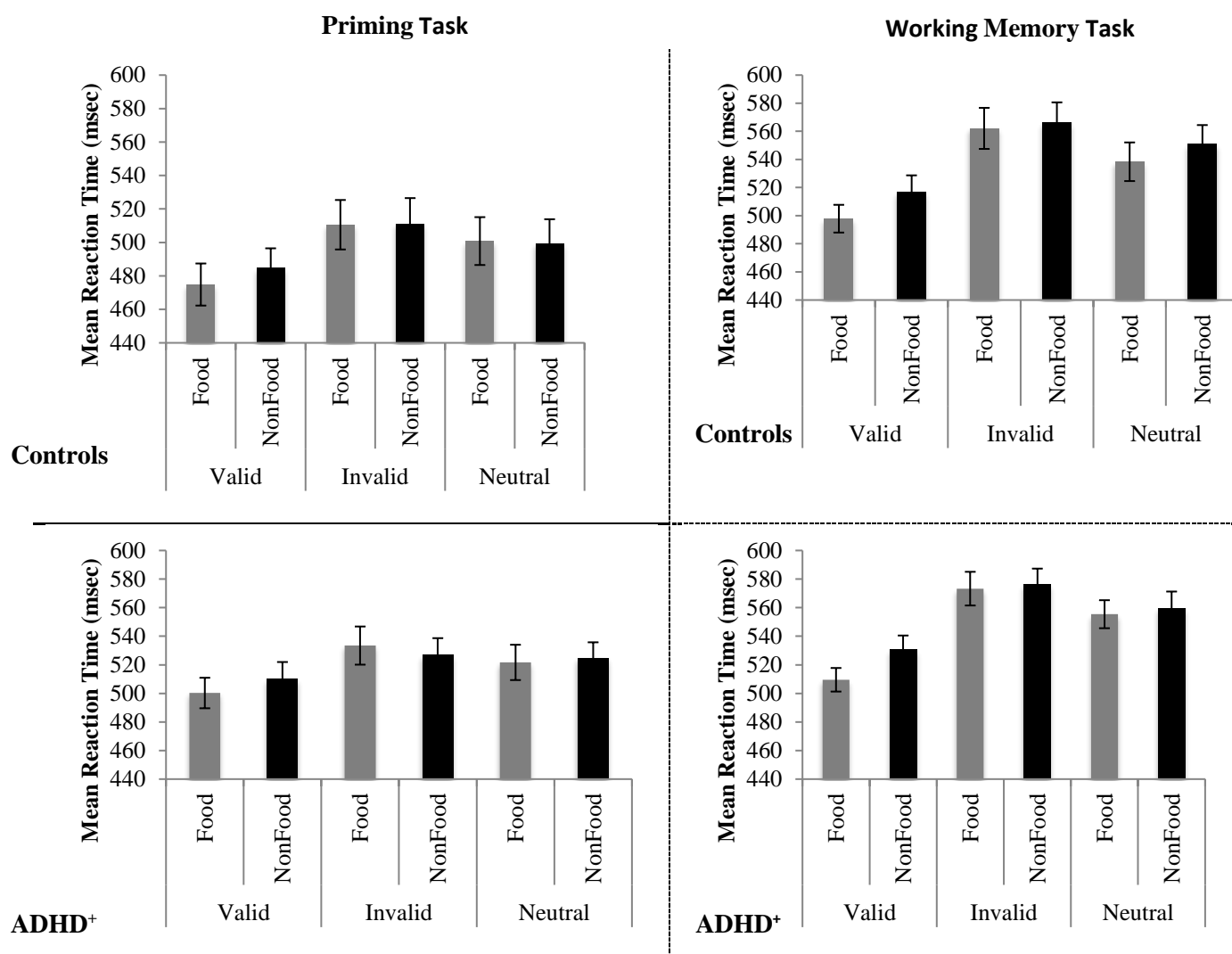


Figure 8. Mean reaction times (in milliseconds) to food, and non-food cues for valid, invalid, and neutral trials, for the priming and working memory task by group. Values are means \pm SEM.

Go/no-go task: Behavioural results.

Responses from five participants could not be used due to technical problems (e.g. responses not recorded during fMRI), therefore behavioural data are presented for 53 participants (25 control; 28 ADHD⁺).

Performance measures on the go/no-go task were analysed independently using a repeated measures ANOVA with stimuli as a within-subjects factor and group as a between-subjects factor. Hunger (average score for the two measures proceeding the scanning session),

BMI and gender are known to affect performance on the go/no-go task and therefore were entered as covariates in the analyses (Loeber, Grosshans, Herpertz, Kiefer, & Herpertz, 2013; Price et al., 2016; Sjöberg & Cole, 2017). No main effect of group ($F(1, 47) = 0.10, p = 0.75, \eta^2 = 0.002$) or any interaction between stimuli and group ($F(3, 141) = 0.61, p = 0.61, \eta^2 = 0.01$) was found on the number of commission errors. Also, there was no main effect of group ($F(1, 47) = 0.01, p = 0.10, \eta^2 = 0.00$) or any interaction between stimuli and group ($F(3, 141) = 0.05, p = 0.98, \eta^2 = 0.001$) on the number of omission errors. Finally, there was no main effect of group ($F(1, 47) = 0.002, p = 0.97, \eta^2 = 0.000$) or interaction between stimuli and group ($F(3, 141) = 2.25, p = 0.09, \eta^2 = 0.05$) on the RT to go stimuli (for group means of performance measures, see Table 4).

Table 4

Descriptive Statistics for the Go/No-Go Task

Performance measure	ADHD ⁺ (n=28)								Controls (n=25)							
	Condition								Condition							
	Go HC		Go LC		Go Sport		Go Toiletry		Go HC		Go LC		Go Sport		Go Toiletry	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
	(Min – Max)		(Min – Max)		(Min – Max)		(Min – Max)		(Min – Max)		(Min – Max)		(Min – Max)		(Min – Max)	
CE	2.11 (0-7)	0.34	1.93 (0-6)	0.27	3.14 (0-10)	0.47	3.29 (0-9)	0.42	2.32 (0-8)	0.42	1.64 (0-6)	0.30	3.32 (0-9)	0.51	2.88 (0-9)	0.45
OE	4.00 (0-23)	1.01	3.54 (0-28)	1.20	3.32 (0-23)	0.97	4.32 (0-35)	1.25	3.92 (0-26)	1.11	3.28 (0-33)	1.31	3.12 (0-17)	0.89	3.96 (0-17)	0.94
RT	495.50 (402.30-567.75)	7.80	484.55 (383.55-591.60)	9.03	484.13 (387.48-604.62)	8.86	500.30 (406.31-578.04)	8.00	501.00 (436.95-588.62)	8.62	475.94 (397.41-558.52)	8.04	474.09 (391.34-539.85)	7.36	502.35 (433.55-558.20)	7.42

CE: Commission errors; OE: Omission errors; RT: Reaction time for correct go trial

Contribution of Specific Symptoms to Disordered Eating

To explore the specific contribution (*if any*) of inattentive and hyperactive/impulsive symptoms of ADHD to measures of disordered eating a moderation analysis was performed, with gender as a moderator, and age and BMI as covariates (for details on covariates see Chapter 4) inattentive and hyperactive/impulsive symptoms of ADHD as predictor variables and the following measures as outcome variables: pasta intake (grams), cookie intake (grams), self-reported measure of binge/disinhibited eating, and self-reported measure of restrictive eating.

Inattentive symptoms and disordered eating.

There was no main effect of inattentive symptoms of ADHD on pasta intake ($b = 2.61$, 95% CI: -3.5 8.7 , $p > 0.05$). However, a significant interaction was observed between inattentive symptoms of ADHD and gender on pasta intake, $b = -14.67$, 95% CI $[-26.80 - 2.55]$, $t = -2.43$, $p = 0.02$. A significant positive relationship was observed between inattentive symptoms of ADHD and pasta intake, $b = 10.33$, 95% CI $[0.57$ $20.09]$, $t = 2.12$, $p = 0.04$ for males, but no for females.

There was a main effect of inattentive symptoms on binge/disinhibited eating as assessed via the self-report measures, $b = 0.85$, 95% CI: 0.35 1.35 , $p = 0.001$, so that increased levels of inattentive symptoms predicted increased binge/disinhibited eating. No other significant main effects and/or interactions between inattentive symptoms of ADHD and gender were observed (95% CI contained zero).

Hyperactive/Impulsive symptoms and disordered eating.

There was a main effect of hyperactive/impulsive symptoms of ADHD on binge/disinhibited eating as assessed via the self-report measures, $b = 1.06$, 95% CI: 0.52 1.62 , $p < 0.001$, so that increased levels of hyperactive/impulsive symptoms predicted

increased binge/disinhibited eating. No other significant main effects and/or interactions between hyperactive/impulsive symptoms of ADHD and gender were observed (95% CI contained zero).

Functional Imaging Data

Effect of go/no-go trials.

A table of main effects, examining activation to No-go versus Go trials, is presented in Table 5a and Figure 9a.

Relative to Go trials, No-go trials were associated with increased activation in bilateral insula, inferior parietal, and middle cingulum, left precuneus and left superior occipital.

We further explored the activation pattern in these regions using a 2 (Go, No-go) by 4 (stimuli type) and 2 (group) mixed ANOVA with individual beta values as the dependent measure in SPSS. None of the regions showed a main effect of group. However, there was a trend for a significant interaction between trial and group in the right inferior parietal ($F(1, 49) = 3.63, p = 0.06, \eta^2 = 0.07$). In this region, both groups showed an increased response across the stimuli type to No-go versus Go trials, but the effect was slightly smaller for the ADHD⁺ ($t(29) = 4.86, p < 0.001, d = 0.89$) compared to the control group ($t(20) = 6.15, p < 0.001, d = 1.34$; see Figure 9a). There was also a trend for a significant interaction between stimuli and group in the left insula ($F(3, 147) = 2.29, p = 0.08, \eta^2 = 0.05$). Across both trial types, the ADHD⁺ group showed enhanced activation compared to the control group in this region in response to HC food items ($U = 203.00, p = 0.03, \eta^2 = 0.09$). Activation in the left insula did not differ between the two groups in response to any other stimuli ($p_s > 0.05$) (see Figure 9a).

The contrast of increased activation of Go relative to No-Go revealed a much smaller number of regions (see Table 5b and Figure 9b), namely, left postcentral gyrus, right caudate

and posterior hippocampus. A marginal main effect of group ($F(1, 49) = 3.57, p = 0.07, \eta^2 = 0.07$) was found in the left postcentral gyrus; across all trials, regardless of stimuli the ADHD⁺ group showed more activation in this region than the control group ($t(49) = 1.89, p = 0.06, d = 0.56$; see Figure 9b).

Table 5

Main Effects: Regions Showing Activation During No-Go and Go Trials Across Subjects Independent of Group (N = 51)

Anatomical Region	Hemisphere	x	y	z	Number of voxels	Z-Peak
a. No-go > Go						
Insula	R	33	18	6	1959	Inf*
Inferior Parietal	R	57	-42	24	4525	7.81*
Middle Cingulum	R	9	18	36	418	7.17*
Insula	L	-27	21	3	759	7.17*
Inferior Parietal	L	-30	-48	39	40 ^{\$}	4.95*
Middle Cingulum	L	-6	-24	27	53	4.75*
Precuneus	L	-15	-69	33	42	4.49
Superior Occipital	L	-27	-72	24	83	4.22
b. Go > No-go						
Postcentral gyrus	L	-33	-27	48	86	Inf*
Caudate	R	21	-9	27	65	5.45*
Posterior Hippocampus	R	36	-42	-3	40 ^{\$}	4.95*

^{\$}Indicates regions where cluster level $p_{FWE} = 0.056$, all other regions were $p_{FWE} < 0.05$ at cluster level. All regions were also reliable at cluster level for $p_{FDR} < 0.05$. * Indicates regions where the peak reliability was $p_{FWE} < 0.05$. L, Left side; R: Right side.

Effect of Food/Non-Food Stimuli

A table of main effects, examining activation to food stimuli versus non-food stimuli, is presented in Table 6 and Figure 10.

Relative to non-food stimuli, food stimuli were associated with increased activation in the right calcarine and left middle occipital. No main effect of group and/or interactions were observed in any of these regions.

No regions were found to be more active in the opposite comparison (non-food>food) at our statistical threshold.

Table 6

Main Effects: Regions Showing Activation in Response to Food Stimuli Relative to Non-Food Stimuli Across Subjects Independent of Group (N = 51)

Anatomical Region	Hemisphere	x	y	z	Number of voxels	Z-Peak
Calcarine	R	15	-90	3	159	6.76*
Middle Occipital	L	-15	-90	-6	138	6.74*

All regions were $p_{FWE} < 0.05$ at cluster level. All regions were also reliable at cluster level for $p_{FDR} < 0.05$. * Indicates regions where the peak reliability was $p_{FWE} < 0.05$. L, Left side; R: Right side.

Effect of HC/LC Food Stimuli

A table of main effects, examining activation to HC food stimuli versus LC food stimuli, is presented in Table 7 and Figure 11.

Relative to LC food stimuli, HC food stimuli were associated with increased activation in the middle occipital, the right parahippocampal, the right caudate, the right frontal inferior operculum extending to anterior insula, the left fusiform and the right middle temporal.

Using the 2 (Go, No-go) by 4 (stimuli type) and 2 (group) mixed ANOVA with individual beta values as the dependent measure, a main effect of group ($F(1, 49) = 9.04$, $p = 0.004$, $\eta^2 = 0.16$) was observed in the right middle temporal, with the ADHD⁺ group showing significantly more activation in this region than the control group ($t(49) = 3.01$, $p = 0.006$, $d = 0.85$). There was also a trend for a significant interaction between trial and group in the same right middle temporal region ($F(1, 49) = 3.20$, $p = 0.08$, $\eta^2 = 0.06$). Activation in this region was marginally greater in the No-go compared to Go trials for the control group ($t(20) = 1.94$, $p = 0.07$, $d = 0.4$), however activation in this region did not differ between the Go and No-go trials for the ADHD⁺ group ($t(29) = 0.21$, $p = 0.83$, $d = 0.0$; see Figure 11).

No regions were found to be more active in the opposite comparison (LC>HC) at our statistical threshold.

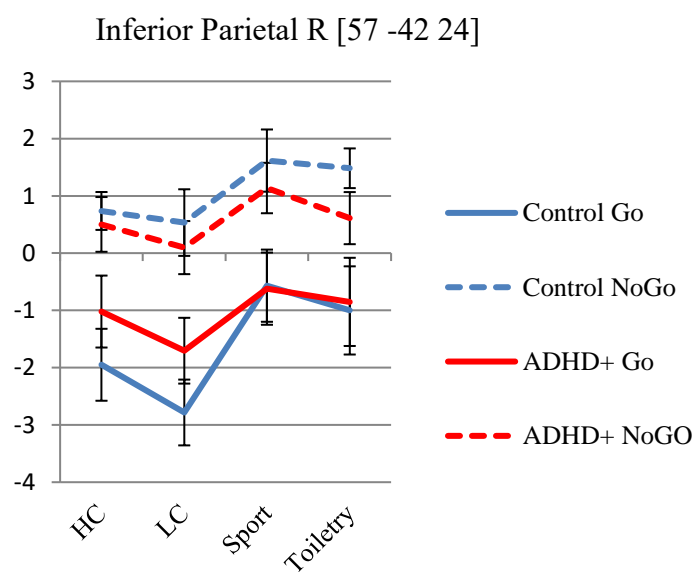
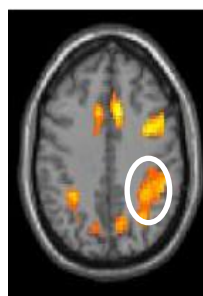
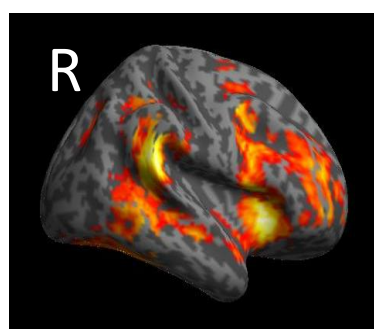
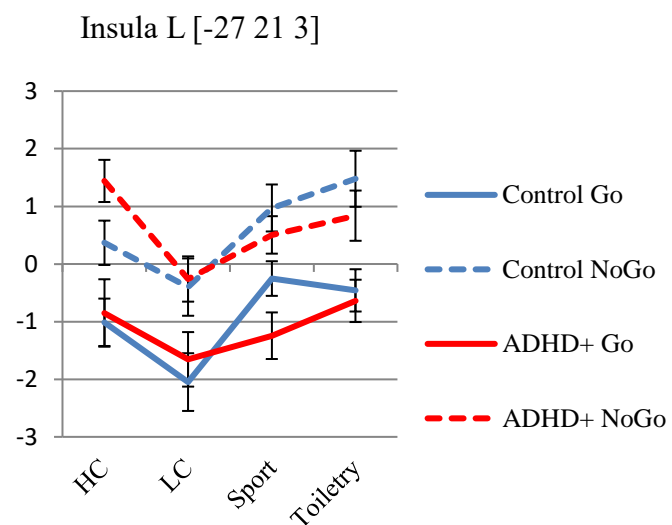
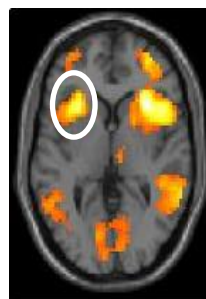
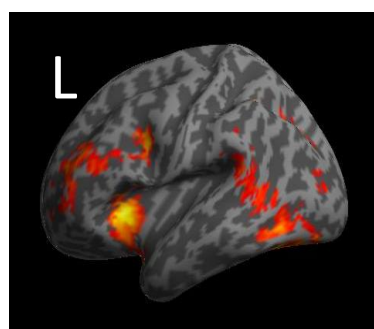
Table 7

Main Effects: Regions Showing Activation in Response to HC Food Stimuli Relative to LC Food Stimuli Across Subjects Independent of Group (N = 51)

Anatomical Region	Hemisphere	x	y	z	Number of voxels	Z-Peak
Middle Occipital	L	-15	-96	3	298	7.17*
Middle Occipital	R	36	-78	15	487	5.82*
Parahippocampal	R	18	-3	-21	434	4.78*
Caudate	R	21	15	6	69	4.35
Frontal inferior operculum	R	45	15	3	66	4.31
Fusiform	L	-27	-45	-12	63	4.02
Middle Temporal	R	48	0	-27	43	3.88

All regions were $p_{FWE} < 0.05$ at cluster level. All regions were also reliable at cluster level for $p_{FDR} < 0.05$. *Indicates regions where the peak reliability was $p_{FWE} < 0.05$. L, Left side; R: Right side.

a. No-go > Go



b. Go > No-go

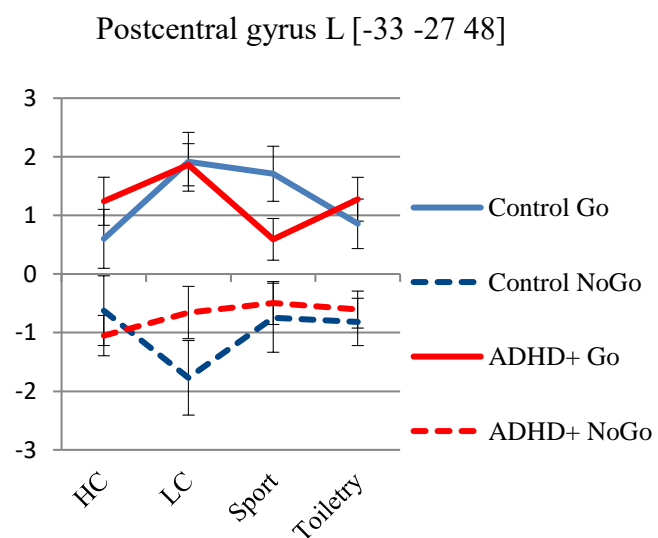
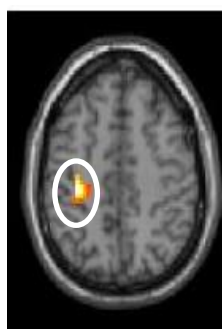
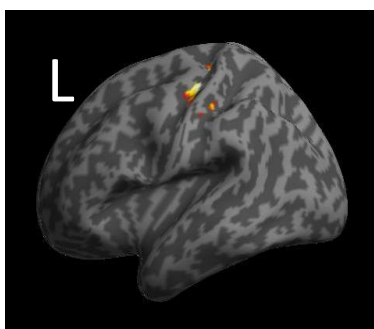
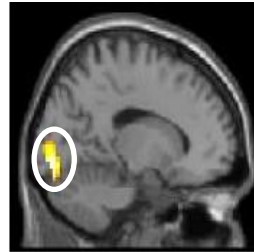
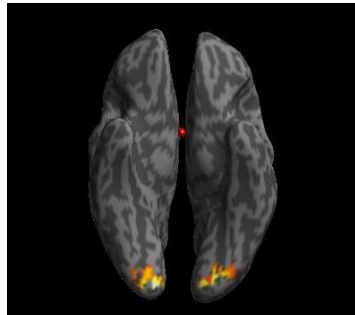


Figure 9. Main effect of Go vs. No-go trials.

Food > Non-Food



Middle Occipital L [-15 -90 -6]

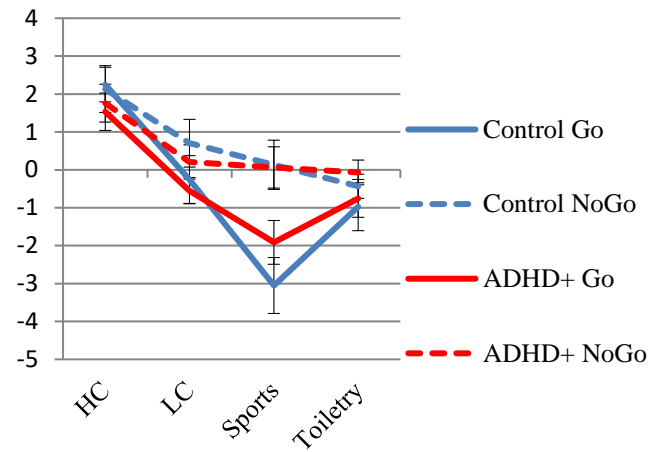
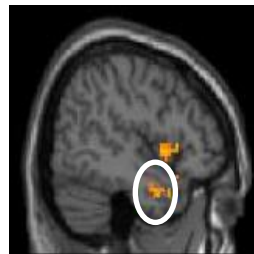
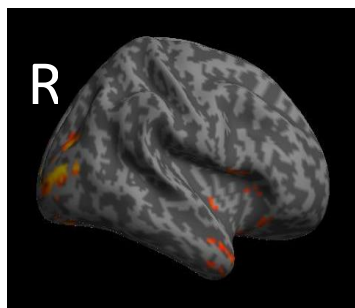


Figure 10. Main effect of Food vs. Non-food stimuli.

HC > LC



Middle Temporal R [48 0 -27]

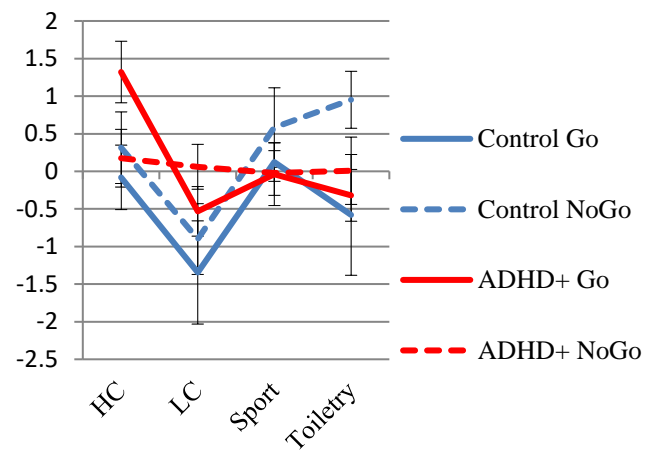


Figure 11. Main effect of HC vs. LC food stimuli.

5.5 Discussion

The present research used an experimental design to investigate eating behaviour in ADHD using laboratory measures, in conjunction with self-report measures, and examined the mechanisms that may underpin the relationship between ADHD and disordered eating behaviour. To the best of our knowledge, this is the first study to assess eating behaviour associated with ADHD using both laboratory and self-report measures, and to investigate the involvement of specific cognitive constructs, such as response inhibition, and neural correlates of eating behaviour, in the same study.

The results demonstrated that there was no main effect of ADHD status on either pasta or cookie intake. However, there was a trend for a significant interaction between gender and group on pasta intake, such that ADHD⁺ females tended to consume less pasta than control females. No significant interaction was observed between gender and group on cookie intake. The pattern of results suggests that ADHD⁺ males tended to consume more pasta and cookies compared to controls, however these differences did not reach significance. However, the significant variability observed in both pasta and cookie intake, suggests that the null findings observed in the present study might be due to insufficient power. To the best of our knowledge, this is the first study to assess eating behaviour in a laboratory setting in adults with ADHD symptomatology and therefore further studies with larger sample sizes are warranted to replicate and extend these initial findings.

Subjective measures of disordered eating indicated that ADHD⁺ subjects scored higher than control subjects on binge/disinhibited eating, and females scored higher than males, although no significant interaction between group and gender was observed. This is in accordance with the literature suggesting a positive association between ADHD and binge/disinhibited eating (Davis et al., 2006; Hudson, Hiripi, Pope, & Kessler, 2007; Strimas

et al., 2008; Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011), and previous reports of higher levels of binge behaviour in female compared to male undergraduate and graduate students (Lipson & Sonnevile, 2017). The absence of any moderating effect of gender on the relationship between ADHD and binge/disinhibited eating is also in agreement with most previous evidence (for a review see Kaisari et al. 2017) and the findings reported in Chapter 4. ADHD⁺ subjects did not differ from controls in restrictive eating behaviour although females scored on average higher than males. Restrictive eating behaviour has rarely been investigated in previous studies, and findings have been inconsistent (see Kaisari et al. 2017). The results from Chapter 4 revealed that ADHD symptomatology was positively associated with restrictive eating behaviour in two independent samples. However, ADHD symptomatology only indirectly predicted restrictive eating behaviour, suggesting that the relationship between ADHD and restrictive eating may be indirect, complex and mediated by other factors. In accordance with our findings, females have been found to report more frequent and higher levels of restrictive eating behaviour compared to males (Davy, Benes, & Driskell, 2006; Jagielska & Kacperska, 2017; Smink, van Hoeken, & Hoek, 2012).

Investigation of the specific contribution (*if any*) of ADHD symptoms on disordered eating revealed a significant positive relationship between inattentive symptoms of ADHD and pasta intake for males, but not for females. In addition, there was a main effect of inattentive symptoms on binge/disinhibited eating as assessed via the self-report measures, such that increased levels of inattentive symptoms predicted increased binge/disinhibited eating. This is consistent with our previous findings, in Chapter 4. Taken together, these findings provide evidence for a specific contribution of inattentive symptoms of ADHD on disordered eating, especially overeating and binge/disinhibited behaviour as assessed both via laboratory measures of food intake and subjective measures of disordered eating. For first

time, we present evidence that gender moderates the relationship between inattentive symptoms of ADHD and overeating behaviour as assessed via pasta intake, such that inattentive symptoms predicted increased pasta consumption for males but not for females. Further research is warranted to replicate these initial findings. In addition, there was a main effect of hyperactive/impulsive symptoms of ADHD on binge/disinhibited eating as assessed via the self-report measures, such that increased levels of hyperactive/impulsive symptoms predicted increased binge/disinhibited eating. These findings are also consistent with our previous results from Study 2 in Chapter 4 where the sample comprised student participants.

Overall, these findings suggest that subjects with high ADHD symptoms show different patterns of eating behaviour compared to control subjects, as assessed both via laboratory and self-report measures. Findings from the questionnaire measures on binge/disinhibited eating were similar to our previous findings reported in Chapter 4, suggesting a significant relationship between ADHD symptomatology and binge/disinhibited eating for both men and women. However, assessment of food intake in the laboratory revealed a different pattern; thus only the inattentive symptoms of ADHD that predicted overeating behaviour, but this relationship was dependent on gender since inattentive symptoms of ADHD predicted pasta intake in men but not in women. The results on restrictive eating behaviour were less clear: there was no significant relationship between ADHD symptomatology and restrictive eating, but women with high ADHD symptoms tended to eat less of the pasta meal in the laboratory. The explanation for this observation is not clear. Given that cookie intake did not differ between ADHD⁺ and control females, it could be suggested that ADHD⁺ females intentionally limited their consumption of the pasta meal, to indulge into the consumption of the highly palatable food later. However, this is only

speculation and future studies need to specifically investigate the potential use of compensatory strategies to control body weight in women with high ADHD symptomatology.

Negative mood differed between ADHD⁺ and control subjects, and ADHD⁺ compared to control individuals reported higher levels of negative mood related both to longer periods of time (anxiety, depression, stress), as well as within shorter periods (within the testing period). Indeed, anxiety and depression are the most common psychiatric comorbidities in ADHD patients (Friedrichs, Igl, Larsson, & Larsson, 2012; Hodgkins, Montejano, Sasane, & Huse, 2011; Kessler et al., 2006), and ADHD symptomatology has been associated with higher levels of self-perceived stress (Hirvikoski, Lindholm, Nordenstrom, Nordstrom, & Lajic, 2009; Lackschewitz, Huther, & Kroner-Herwig, 2008).

The current study adds to the literature, providing evidence for stronger intensity of negative states of mood among ADHD⁺ individuals even within shorter periods of time (within the testing period), although reports related to positive states of mood (happy, energetic and alert) as assessed within the testing day were not found to differ between ADHD⁺ and control individuals. Although ADHD⁺ subjects scored higher on negative states of mood throughout the testing day, at the end of the testing session their ratings did not differ from those of controls. The preceding consumption of food might have resulted in a decrease of negative states of mood in ADHD⁺ subjects. Indeed, food is known for its mood enhancing properties (Gibson, 2006). However, this is only speculation and future studies are needed to test whether experimentally manipulating mood status can have an impact of food intake in participants with ADHD, and change perception of mood because of food consumption.

Accuracy in detection of internal bodily sensations, as measured by the heartbeat perception task was not found to differ between ADHD⁺ and control individuals. Findings from Study 2, Chapter 4 also did not support an association between core symptoms of

ADHD and interoceptive sensitivity as assessed via the heartbeat task. Taken together, these findings suggest that further studies are required to investigate the use of a heartbeat task as an index of interoceptive sensitivity in individuals with ADHD symptomatology.

Similarly, no difference was observed in awareness and/or reliance on internal/hunger satiety signals between ADHD⁺ and control subjects in this study. Findings from Chapter 4, showed that inattentive symptoms of ADHD were associated with decreased awareness of internal signs of hunger and satiety, and in turn these deficits were positively associated with disordered eating, especially binge/disinhibited eating. However, in the present study differences are presented only at a group level, and subjects are categorised as controls or ADHD⁺ based on total ADHD symptoms. It is possible that the relationship between awareness and/or reliance on internal/hunger satiety signals and ADHD symptoms may be driven specifically by the inattentive symptoms of ADHD.

Attentional biases toward food cues did not differ between ADHD⁺ and control subjects. The findings from Chapter 2 suggested that overweight/obesity status has a significant impact on attentional biases toward food cues, such that when participants were asked to hold a food cue in working memory they had more difficulty to disengage from that cue in the subsequent selection task, even if this was irrelevant to the task, if they were overweight/obese compared to the controls. In the current study, most participants were categorized as normal-weight. Future studies with adequate numbers of overweight/obese individuals are warranted to examine whether the effect of ADHD on attentional biases to food cues (*if any*) is dependent upon overweight/obesity status.

At the behavioural level, ADHD⁺ subjects did not differ from controls in the mean number of commission errors, omission errors and/or RT to go stimuli, as assessed during the go/no go task, both during the neutral- based and the food-based task. Previous research has

found that auditory stimulation improves ADHD participants' performance on several tasks (Abikoff, Courtney, Szeibel, & Koplewicz, 1996; Soderlund, Sikstrom, & Smart, 2007) whilst not affecting the performance of a control group. Therefore, it is possible that the result might have been different if participants were asked to complete the go/no go task outside the scanner. However, another possibility is that ADHD⁺ subjects use alternative strategies to compensate for deficits. Indeed, impulsivity as assessed subjectively via a self-report measure was found to differ between ADHD⁺ and controls in the present study; ADHD⁺ subjects scored higher than controls on all subscales of impulsivity (attentional, motor, non-planning), but the effect was stronger for the attentional subscale. Similarly, Dillo et al. (2010) found no difference in performance during a go/no go task between adults with and without ADHD. In addition, adults with ADHD showed enhanced activity in the parietal cortex, which is known to play an important role in attention, suggesting that adults with ADHD can compensate for their deficits for a short time because of parietal hyperactivation.

Our findings support this hypothesis, as the right inferior parietal showed a higher response during No-go than Go trials in both groups. However, relative to controls, the ADHD⁺ participants showed enhanced activation in this region during the Go trials, with a smaller difference in response to Go versus No-go. ADHD⁺ compared to control participants also showed increased activation in the left postcentral gyrus, across all trial types, regardless of stimuli. Enhanced activation in this region, among others, has also been reported in children with ADHD during a go/no go task (Ma et al., 2012), and has been suggested to represent compensatory brain activation to sustain normal response inhibition function. Furthermore, activation in the right middle temporal, was stronger for the ADHD⁺ than the control group. The ADHD⁺ group also showed a similar level of response of the right middle temporal to the Go and No-go trials, while controls primarily recruited this region during the

No-go trials. Superior and middle temporal regions are involved in mental preparation (Kounios et al., 2006; Tian et al., 2011) recalling semantic rules necessary for task completion (Simmons & Martin, 2009; Simmons, Reddish, Bellgowan, & Martin, 2010).

Thus, the enhanced activation in the right middle temporal observed in ADHD⁺ compared to controls, is suggestive of preparedness and attention to the task. Finally, ADHD⁺ compared to control participants showed increased activation in the left insula in response to HC food items, across both trial types (Go and No-go trials). The latter could suggest increase vigilance specifically in the context of HC food. The insula plays a role in encoding the reward value of food (Batterink et al., 2010; Stice et al., 2008; Stoeckel et al., 2008). Thus, our finding of greater activation in this food reward region in ADHD⁺ subjects in response to HC food items suggests that, across all trial types (Go and No-go trials), the images of HC food items may have been more attractive to ADHD⁺ individuals compared to controls.

Across both groups, relative to non-food stimuli, food stimuli were associated with increased activation in the right calcarine and left middle occipital, areas mainly involved in visual processing (Grill-Spector, Kourtzi, & Kanwisher, 2001; Yang, Deng, Xing, Xia, & Li, 2015). In a meta-analysis on the neural correlates of processing visual food cues, the lateral occipital cortex was consistently found to be activated in response to viewing food pictures (van der Laan, de Ridder, Viergever, & Smeets, 2011). This increased activation in visual processing areas in response to food versus non-food stimuli is unlikely to be explained by a difference in visual characteristics because the pictures were matched for general visual characteristics. Furthermore, van der Laan et al. (2011) also reported that in the majority of studies that compared food and non-food stimuli, the stimuli were matched for visual characteristics (e.g. colour, luminance, visual complexity). An alternative explanation for why food pictures elicit a stronger activation in visual processing areas is that emotionally salient

stimuli like food lead to heightened attention and therefore more extensive visual processing (Killgore & Yurgelun-Todd, 2007). No main effect of group and/or any interactions were observed in these regions, suggesting that food compared to non-food pictures attracted attention equally for controls and ADHD⁺ participants. Furthermore, across both groups, HC food stimuli was associated with increased activation in the middle occipital, the right parahippocampal, the right caudate, the right frontal inferior operculum extending to anterior insula, the left fusiform and the right middle temporal, areas typically involved in motivational salience and mental preparation (Kounios et al., 2006; Pursey et al., 2014; Tian et al., 2011).

Our study has several strengths and limitations. The sample size was selected using power calculations for brain activation patterns in regions relevant to response inhibition and it is among the largest in the relevant literature comprising fMRI studies of response inhibition (Hart et al., 2013). Both males and females were included to allow investigation of any gender-specific effects. The experimental conditions were designed to match those of everyday life, as participants were asked to consume their typical breakfast before coming to our research facilities, and lunch was offered around their usual lunch time, followed by a snack food. However, it is worth noting, that despite this relatively large sample size, the observed impact of ADHD on eating behaviour and responses to food stimuli were small. The large variability within the population of ADHD and control participants in response to food might have masked specific effects of ADHD. As we only included a sub-clinical population of ADHD, it is also possible that these effects would have been larger in a clinical sample.

Our sample is not representative of the general population. The participants were all young, highly educated University students; thus, the results cannot be generalised to other population groups. Finally, as the present research was not specifically designed to assess the

involvement of core symptoms of ADHD on disordered eating, the relevant results cannot be considered as conclusive and future studies are needed to specifically assess the role of inattentive and hyperactive/impulsive symptoms on disordered eating, and validate these initial findings.

In conclusion, the present results showed that participants with high ADHD symptoms show different patterns of eating behaviour, as assessed both via laboratory and self-report measures. Inattentive symptoms of ADHD were found to directly predict binge/disinhibited eating behaviour, and for first time we provide evidence that inattentive symptoms of ADHD predicted overeating behaviour, as assessed by consumption of a pasta meal in a laboratory setting. This relationship was dependent upon gender, as only in males inattentive symptoms of ADHD predicted pasta intake. Attentional processing to food cues was not found to differ between controls and individuals with high ADHD symptoms. Furthermore, although subjects with high ADHD symptoms reported higher levels of impulsivity on self-report measures, analysis of responses to the go/no go task is suggestive of compensatory brain activation in adults with high ADHD symptoms to sustain normal response inhibition function. However, neural response to food cues, specifically in the insula to HC food items, indicated potential reward-related differences between controls and individuals with high ADHD symptoms. Further, research is required to validate these initial findings.

Chapter 6: General Discussion

6.1 Introduction

The overall aim of this thesis was to better understand the specific cognitive and neural mechanisms that may serve as risk factors for the development of disordered eating behaviour. Specifically, the research presented here was conducted in order to answer the following research questions: (1) Does attentional processing of food cues distinguish overweight/obese from normal-weight individuals? (2) Can differential processing of food cues predict disordered eating behaviour as assessed in a laboratory setting, and future weight gain at one-year follow up? (3) What is the evidence for an association between ADHD and disordered eating? (4) Is there evidence to suggest an association between ADHD and specific types of disordered eating behaviour? (5) Are core symptoms of ADHD differentially associated with specific types of disordered eating behaviour? (6) Are there factors that affect the direction and/or strength of any relationship between ADHD symptoms and disordered eating (moderators)? (7) Which factors could explain the relationship between ADHD symptoms and disordered eating behaviour (mediators)? (8) Does eating behaviour as assessed both via laboratory and questionnaire measures differ between controls and individuals with high ADHD symptomatology (9) Are there differences in the neural processing of food cues that could explain disordered eating behaviour in individuals with high ADHD symptomatology? Each research question was addressed and the findings are summarised here, followed by a discussion of strengths and limitations of the studies, together with suggested future directions for research. The clinical implications of findings are also considered before adding some concluding remarks.

6.2 Overview of Findings

Chapter 2 presented evidence on reward-related differences in responses to food cues between normal-weight and overweight/obese individuals using a paradigm that allowed for the first time assessment of both automatic (bottom-up) and top-down guidance of attention via working memory. Although, high-responsiveness to food cues (attentional bias) has been hypothesised to be linked to overeating and weight gain, the evidence to date is mixed. In a sample of 43 overweight/obese individuals and 49 healthy controls attentional processing of food cues distinguished overweight/obese individuals from normal-weight controls, but only when food related information was manipulated in working memory, similar to food preoccupation that many people with obesity report (Israel, Stolmaker, & Andrian, 1985; Lynch, Eppers, & Sherrodd, 2004). Specifically, when participants were asked to hold food-related information in working memory, overweight/obese compared to normal-weight subjects were much slower to detect the target in the invalid trials where the cued item matched the distractor compared to valid trials where the cued items matched the target. These findings indicate that where food cues are available and match representations in working memory, overweight/obese individuals find it much harder to disengage from those cues even if they are irrelevant to current goals (e.g. target selection). These data provide the first evidence for a novel mechanism underlying attentional biases for food cues in overweight/obese individuals through working memory, even under conditions of satiety. We also found that overweight/obese individuals showed a different pattern of eating behaviour as assessed during a bogus taste test compared to normal-weight individuals. Thus, overweight/obese individuals consumed more energy from highly palatable foods than normal-weight individuals, while no difference was observed in the consumption of fruits and vegetables. These differences in food intake could not be explained by differences in

attentional processing of food cues, suggesting that the relationship between attentional biases and food intake might be complex. However, differences in attentional processing of food cues, when food-related information was held in working memory predicted weight gain at one-year follow-up, after taking into account other potential predictors of BMI change, highlighting the potential importance of top-down mechanisms in the regulation of body weight.

In Chapter 3, the evidence for an association between ADHD and disordered eating was assessed in a systematic review. A dimensional approach to the study of ADHD and disordered eating was used, which allowed a large number of studies to be systematically evaluated. Specifically, 75 studies were included in the review, and findings revealed that overall there is a moderate strength of evidence for a positive association between ADHD and disordered eating, and with specific types of disordered-eating behaviour, in particular, overeating. In contrast, evidence for an association between ADHD and restrictive eating behaviour is more limited, and the findings are mixed. A point of note was that most of the studies were not specifically designed to assess the association between ADHD and disordered eating, resulting in suboptimal study designs in many cases, with the potential for biased results. Furthermore, mechanism testing was very limited and the specific contribution (*if any*) of core symptoms of ADHD to the development and/or maintenance of disordered eating behaviour was unclear.

In Chapter 4, the studies were specifically designed to investigate potential associations between core symptoms of ADHD (inattention and hyperactivity/impulsivity) and disordered eating, including both binge/disinhibited eating and restrictive eating behaviour, and to assess potential mediational pathways of association. Specifically, it was tested whether negative mood, a composite index reflecting anxiety, depression and stress,

and/or deficits in awareness and reliance on internal hunger/satiety cues mediated any association between ADHD and disordered eating behaviour, while controlling for important confounds identified in a systematic review of previous research (see Chapter 3). In addition, the potential moderating effects of age, gender, BMI and/or ADHD medication in relationships between core symptoms of ADHD and disordered eating were investigated. In two independent studies, it was found that core symptoms of ADHD positively correlated with both binge/disinhibited and restrictive eating and negative mood mediated the relationships. Deficits in awareness and reliance on internal hunger/satiety signals also mediated the association between inattentive symptoms of ADHD and disordered eating, especially binge/disinhibited eating. There was no evidence of any moderation effects. These findings provide the first evidence for a direct relationship between inattentive symptoms of ADHD and binge/disinhibited eating behaviour.

The study presented in Chapter 5, aimed to further build on findings from Chapter 4: differences in eating behaviour between individuals with high ADHD symptoms and control individuals were assessed using laboratory measures of eating behaviour in conjunction with questionnaire measures. An experimental procedure was used that allowed investigation of eating behaviour both in the context of a typical meal (lunch), but also in the absence of hunger (snack intake), providing a potential behavioural measure of binge-like eating behaviour. The involvement of specific cognitive constructs (e.g. inhibition) and neural correlates of eating behaviour was also assessed using fMRI. Participants with high ADHD symptoms showed different patterns of eating behaviour, as assessed both via laboratory and self-report measures. More specifically, findings from the questionnaire measures on binge/disinhibited eating were similar to the findings reported in Chapter 4, suggesting a significant relationship between ADHD symptomatology and binge/disinhibited eating for

both men and women. However, assessment of food intake in the laboratory revealed a different pattern, as only the inattentive symptoms of ADHD predicted overeating behaviour, but this relationship was dependent upon gender since inattentive symptoms of ADHD predicted pasta intake in men but not in women. The results on restrictive eating behaviour were less clear. Thus, there was no significant relationship between ADHD symptomatology and restrictive eating, but women with high ADHD symptoms tended to eat less of the pasta meal in the laboratory. At the behavioural level, attentional processing to food cues as well as inhibitory control both in the context of food and non-food cues were not found to differ between controls and individuals with high ADHD symptoms. However, neural response to food cues, especially the insula response to HC food items, indicated reward-related differences between controls and individuals with high ADHD symptoms. There was also evidence for compensatory brain activation in adults with high ADHD symptoms that were interpreted as reflecting a compensatory response to sustain normal response inhibition.

6.3 Theoretical and Clinical Implications

The findings presented within this thesis may have important theoretical and clinical implications. Eating is a complex behaviour, and throughout this thesis emphasis has been placed on the role of specific cognitive factors in influencing eating behaviour. The results suggest an important role of attention in eating behaviour, and to better understand how attention can guide and/or directly influence eating behaviour, the data are discussed in relation to the role of attention in food cue processing before an eating episode starts and during intervals between eating episodes.

The Role of Attention in Food Cue Processing Prior to the Start of an Eating Episode. Food cues may trigger a desire to eat in two ways, by the sight of food and by preoccupation with food thoughts, especially if one is hungry (Berry, Andrade, & May, 2007).

However, individual differences have been reported in responsiveness to food cues (Fedoroff, Polivy, & Herman, 1997), such that for certain individuals it might be more difficult to ignore the presence of food cues around them or they may be more often preoccupied with food thoughts. Holding food related information in working memory has been found to guide attention towards food-related stimuli in the environment (Higgs, Rutters, Thomas, Naish, & Humphreys, 2012; Rutters, Kumar, Higgs, & Humphreys, 2015). The present findings suggest that attentional processing of food cues can distinguish overweight/obese from normal-weight individuals. Thus, overweight/obese individuals find it more difficult to disengage from a food cue in the environment, when they hold-food related information in working memory, even if this is irrelevant to their current goals (e.g. target selection) highlighting the importance of working memory in guiding attention to food cues. Attentional bias to food cues can drive the initiation of an eating episode.

Although, the underlying mechanisms are unclear one possibility is that paying attention to a stimulus increases the readiness to execute actions associated with that stimulus, for example reaching for a tempting food (Krebs, Boehler, & Woldorff, 2010). Another possibility is that selective attention to sensory/hedonic attributes of food biases choice towards food consumption because these attributes of food are weighed more strongly than longer-term goals in reward evaluation processes (Werthmann, Jansen, & Roefs, 2016). However, investigation of the effect of attentional bias on food intake in a laboratory setting is limited and has yielded mixed findings (Nijs, Muris, Euser, & Franken, 2010; Werthmann et al., 2014; Werthmann, Roefs, Nederkoorn, & Jansen, 2013; Werthmann et al., 2011). The findings of the present thesis do not support a significant relationship between attentional processing of food cues and food intake, although differential attentional processing was found to be a predictor of future weight gain. Therefore, it is possible that longer-term goals

(e.g. weight-loss or adoption of a healthy eating pattern) may moderate the relationship between attentional bias and food intake in the short term.

The Role of Attention in Intervals between Eating Episodes. Evidence from the present research suggest a role for inattentive symptoms of ADHD in binge/disinhibited eating behaviour, and overeating behaviour assessed by laboratory measures of food intake, especially for males. Attention paid to food during an eating episode has previously been found to affect subsequent food consumption. For example, attentive eating has been found to decrease subsequent snack intake by augmentation of encoding of food memories (Higgs & Donohoe, 2011; Robinson, Kersbergen, & Higgs, 2014). On the other hand, there is evidence that if encoding of episodic food memories is disrupted (e.g. by watching TV or playing a computer game) while eating, subsequent snack intake is increased (Higgs, 2016; Higgs & Woodward, 2009; Mittal, Stevenson, Oaten, & Miller, 2011; Oldham-Cooper, Hardman, Nicoll, Rogers, & Brunstrom, 2011). These findings suggest that the observed link between inattentive symptoms of ADHD and binge/disinhibited and/or overeating behaviour could be explained by degraded memory encoding during an eating episode. It is possible that individuals with prominent inattentive symptoms of ADHD are easily distracted while eating, thereby encoding inadequate memories of recent eating, which may result in increased food consumption during a subsequent eating episode. There is also evidence to suggest that remembered food intake is a better predictor of subsequent hunger than the amount eaten (Brunstrom et al., 2012). In contrast with the findings in overweight/obese individuals, individuals with high ADHD symptoms compared to controls were not found to differ in attentional processing of food cues, when food-related information was manipulated in working memory.

Taken together these findings suggest that while attentional bias to food cues might trigger initiation of eating in overweight/obese individuals, providing a behavioural risk factor for overeating and weight gain, this does not seem to explain disordered eating behaviour in individuals with high ADHD symptoms. The findings from fMRI studies presented in Chapter 5 also support this notion, since although food compared to non-food cues were associated with increased activation in visual processing areas, suggested to be enhanced by attention, no difference in activation was observed between controls and individuals with high ADHD symptoms. Therefore, it is proposed that attentional deficits in ADHD may negatively impact memory encoding during an eating episode, leading to increased food intake on subsequent eating occasions.

From a clinical perspective, the predictive value of differential attentional processing of food cues on weight gain when food related information was held in working memory may suggest that novel treatments for the management of overweight/obesity could be developed. Specifically treatments could be designed to influence top-down guidance of attention from working memory, resulting in reduced attentional bias for food cues. Furthermore, it would be of interest to assess whether attentional processing of food cues when food related information is held in working memory can predict treatment response in individuals participating in weight-loss programs. This could provide a behavioural measure of potential outcome allowing clinicians involved in weight management to identify treatment ‘resistant’ individuals at an early stage and enabling more personalised treatment.

Throughout this thesis consistent evidence is presented for an association between ADHD symptomatology and disordered eating. Disordered eating is a risk factor for full-syndrome eating disorders (Goldschmidt, Aspen, Sinton, Tanofsky-Kraff, & Wilfley, 2008), which can significantly impact both the physical and the psychological health of individuals.

Therefore, it is important that clinicians involved in the management of ADHD also monitor their patients for the presence and/or development of EDs and/or disordered eating behaviours. This is particularly important for children, as any information relevant to their eating behaviour is provided mainly by parents and/or caregivers. Increased awareness of the associated risks for individuals with ADHD to develop disordered eating behaviour could have a significant public health impact, allowing detection of eating disorders in their infancy when therapeutic treatments are associated with better outcomes (BEAT, 2015). In addition, evidence from the present research suggests that prevention and intervention programs for eating disorders would be likely to benefit from treating mood disorders and interoception deficits in individuals who score highly on ADHD symptomatology.

The results provide novel evidence for a significant association between inattentive symptoms of ADHD and binge/disinhibited eating behaviour, as assessed via self-report measures, and overeating behaviour (particularly for males), as assessed via laboratory measure of food intake. These findings suggest that treatments and behavioural therapies that directly target attentional deficits may be particularly effective in the management of both ADHD and overeating and/or binge eating behaviour.

Finally, our findings of a possible association between ADHD and restrictive eating behaviour question whether the recently approved drug for the treatment of moderate to severe BED, lisdexamfetamine (US Food and Drug Administration, 2015) would be an effective therapy for individuals with ADHD who exhibit restrictive eating. It is proposed that assessment of eating behaviours should be an important factor to consider when making treatment decisions.

6.4 Strengths, Limitations and Future Research

The research presented in the context of the present thesis has a number of strengths. First, to investigate the role of core cognitive processes involved in regulation of eating behaviour we moved beyond the study of normal-weight individuals and recruited individuals with overweight/obesity, in addition to individuals with well-established impairments in core cognitive processes, such as attention and inhibitory control, and individuals with high ADHD symptomatology. In addition, particular emphasis was placed on recruitment of both male and female participants. Although, studying lean individuals can provide a starting point to elucidate the role of specific cognitive constructs on appetite and food intake regulation in obesity and ADHD, it is of paramount importance to investigate these proposed mechanisms in individuals with obesity and symptoms of ADHD. Findings from lean individuals do not necessarily translate to overweight/obese individuals, and therefore studies with a between-subjects design are better suited to assess differences in cognitive processes (e.g. attentional processing) that may underpin differences in eating behaviour. To date, few studies have investigated attentional processing of food cues in individuals with overweight/obesity (for reviews see Hendrikse et al., 2015; Werthmann, Jansen, & Roefs, 2015), and to the best of our knowledge our sample of overweight/obese individuals is the largest reported in the relevant literature. Given that attentional processing of food cues might differ between overweight and obese individuals, future research should study overweight and obese individuals separately.

The recruitment of individuals with high ADHD symptomatology from non-clinical settings (often referred to as an intermediate phenotype) enables mechanism testing of core symptoms of ADHD, and disordered eating behaviour, while avoiding potential confounds associated with clinical research in patient populations receiving medication for the disorder. For example, it is known that drug therapies for ADHD such as methylphenidate can have a

pronounced effect on appetite (Findling et al., 2008). Therefore, before moving to clinical research in patients, that may involve complex interactions between ADHD diagnosis, drug and/or other treatment medications, and other comorbid disorders, it is valuable to establish relationships between core symptoms of ADHD and disordered eating using an intermediate phenotype approach. In the studies presented within this thesis, we provide consistent evidence of a role for the core symptoms of ADHD in disordered eating behaviour, as for the mediational pathways of association. The present research could be extended to patients including younger individuals diagnosed with ADHD. This is particularly important given the higher associated risk for younger individuals to develop EDs and/or disordered eating behaviours.

Throughout this thesis, particular emphasis has been placed on the assessment of eating behaviour through the use of rigorous methods. In the study presented in Chapter 2, food intake was assessed via a means of bogus taste test. In a recent participant level analysis of 31 published laboratory studies that used the taste test to measure food intake, the results indicated that the bogus taste test is a valid measure of laboratory food intake (Robinson et al., 2017). Existing research assessing the relationship between attentional biases to food cues in overweight/obesity and food intake has been limited to the investigation of energy-dense highly palatable food items (Nijs et al., 2010; Werthmann et al., 2011). However, to test whether the effect (if any) of differential attentional processing to food cues in food intake is specific to energy-dense highly palatable food cues, a direct comparison between ‘‘healthy’’ and ‘‘unhealthy’’ food items is required. To the best of our knowledge, the Study presented in Chapter 2 is the first to investigate potential differences in eating preferences between normal-weight and overweight/obese individuals that could be explained by differences in attentional processing, using both a range of ‘‘healthy’’ and ‘‘unhealthy’’ food items during a bogus taste

test. Furthermore, given that hunger and eating styles (e.g. dietary restraint) can have a significant impact on food intake (Robinson et al., 2017), to ensure that any effects are specific to differential attentional processing of food cues, all participants were asked to come to our research facilities after an overnight fast and were offered a standard meal for consistency. Eating styles were also taken into account to the model of food intake. In the studies presented in Chapter 4, a number of validated questionnaires were used to assess disordered eating behaviour. As noted in Chapter 3 in the findings of the systematic review, previous research has mainly focused on the assessment of overeating behaviours and their relation to ADHD and/or ADHD symptomatology. To address this issue, in the research conducted within the present thesis we used validated questionnaires to assess both binge/disinhibited eating and restrictive eating behaviour. Self-report measures of eating behaviour are useful tools to assess eating styles and behaviours, but are subject to individual interpretations, since certain questions may be not be interpreted in the same way by all individuals (Tury, Gulec, & Kohls, 2010). Therefore, it is important that experimental research combines both self-report and laboratory measures of eating behaviour. Building upon findings from Chapter 4, in Chapter 5, laboratory measures of food intake were used in conjunction with well-validated self-report measures. To match normal eating outside the laboratory, participants consumed a lunch (pasta meal) around their usual lunch time until satiated, followed 20 minutes later by eating as much as they wanted of a palatable cookie snack to investigate eating in the absence of hunger. Analysis of the pattern of the results showed that individuals with high ADHD symptomatology, especially males, tended to consume more both of the pasta meal and the cookie snack, although these differences were not significant. The great individual variability observed in food intake, suggests that these apparent null findings reported in Chapter 5 might be due to lack of power and future studies

with bigger sample sizes are warranted to replicate and extend these initial findings. This approach could also be used to study eating behaviour in other mental disorders associated with disordered eating such as schizophrenia and depression (see Kaisari et al., 2007).

Our approach to the study of ADHD and disordered eating is consistent with the National Institute of Mental Health Research Domain Criteria Initiative (RDoC) guidelines, which encourages research on dimensions of observable behaviour and neurobiology rather than a categorical, symptom-based approach to the study of mental health (Insel et al., 2010). However, it is important to note that at present there are no recommendations in RDoC on how to measure and/or assess eating behaviour using objective and/or self-report measures. Our findings suggest that laboratory measures of food intake should be used in conjunction with self-report measures to better capture the spectrum of disordered eating behaviours. Our model of laboratory assessment of food intake can provide a useful tool to study differential patterns of eating behaviour depending upon the type of food (standard meal vs. highly palatable food item).

Performance based tasks were also used to assess the role of specific cognitive constructs on eating behaviour. The paradigm that was employed to assess attentional processes to food cues, has a significant advantage compared to paradigms commonly used in the field, as it enables investigation of both bottom-up processes and top-down processes in guidance of attention. The results provide the first evidence that participants who show enhanced attentional biases for food cues when holding food-related information in working memory, are at increased risk for future weight gain. An important question for future research is to assess whether the efficacy of treatments for overweight/obesity (e.g. behavioural or pharmacological interventions) depends upon the extent to which they

influence top-down guidance of attention from working memory, resulting in reduced attentional bias for food cues.

To investigate the specific influence of appetite food cues on self-control, a food go/no go task, using pictures of low- and high-calorie food stimuli along with neutral-based stimuli was developed. This design enables investigation of the extent to which inhibitory control deficits are driven by food and more specifically certain types of food (e.g. highly palatable foods) that are commonly associated with overeating behaviour. The findings suggest that individuals with high ADHD symptomatology may use compensatory mechanisms to sustain normal response inhibition. Future research is required to validate this task in different populations (e.g. overweight/obese individuals) and to confirm its utility to measure food-specific response inhibition.

Despite these strengths, certain limitations should be considered. Firstly, although assessment of food intake in the laboratory has been designed to mimic naturalistic settings, the issue of ecological validity remains. In addition, it is possible that participants' eating behaviour when assessed in the laboratory is influenced by the presence of the researcher (Robinson, Kersbergen, Brunstrom, & Field, 2014; Roth, Herman, Polivy, & Pliner, 2001). Thus individuals might alter their eating behaviour to avoid judgement, which can result in social desirability bias (Goldman, Herman, & Polivy, 1991; Robinson, Kersbergen, Brunstrom, et al., 2014; Vartanian, 2015; Vartanian, Herman, & Polivy, 2007). Although, in the studies presented within the present thesis this source of bias was minimised, as participants were left alone in a room to complete the food intake assessments, the potential of a social desirability bias cannot be excluded.

Another source of bias relates to the recruitment of a relatively homogenous group of highly educated participants; most of the participants that took part in the laboratory-based

studies presented in the present thesis were University students, and the sample that took part in the online study generally comprised highly educated adults. Therefore, future studies are warranted to replicate and extend the current findings to more representative population samples. Furthermore, despite the fact that all studies were advertised so that both males and females could take part, generally males comprised a smaller proportion of the sample, and only in the study presented in chapter 5, where pre-screening proceeded actual testing, did the sample comprise approximately equal numbers of males and females. Thus, future studies should extend the findings presented within this thesis to more equally distributed samples of males and females.

Another potential limitation that should be considered relates to the use of a self-report measure to assess ADHD. Although, the CAARS-S: SV is a well-validated measure to assess current ADHD symptoms, it would be valuable for future research to incorporate performance-based measures to assess core symptoms of ADHD. In the studies presented in Chapter 4, an attempt was made to examine the specific contributions of hyperactive and impulsive symptoms of ADHD to disordered eating. However, any conclusions should be tempered, as no objective measure of hyperactivity was used. The QbTest is an objective test that could be used in future research to evaluate the core symptoms of ADHD (hyperactivity, inattention and impulsivity) (see <https://www.qbtech.com/>), enabling more definitive conclusions to be drawn concerning their involvement in disordered eating. It is also important to note that although the findings from Chapter 4 suggest that the core symptoms of ADHD relate both to binge/disinhibited and restrictive eating pathology, the cross-sectional nature of the research does not allow us to conclude whether binge/disinhibited eating and restrictive eating characterise two distinct groups within our sample or core symptoms of ADHD predispose individuals to increased eating pathology, with cycling between bingeing

and restrictive eating. Longitudinal studies are needed to understand the complex interaction that may arise due to cyclical shifting between different subtypes of EDs (Fairburn & Harrison, 2003) and the change of predominant symptoms of ADHD that may occur throughout the lifetime (Holbrook et al., 2016).

Although, future research directions have been outlined in a framework to support the management of the limitations discussed, findings from the present thesis suggest as a research priority the investigation of the underlying mechanisms through which inattentive symptoms of ADHD can drive overeating behaviour. The suggestion that inattentive symptoms may result in suboptimal memory encoding during an eating episode, leading to subsequent increases in food intake needs to be formally investigated. For example, individuals with high and low ADHD symptoms could be recruited and asked to consume a meal in a laboratory setting (*ad libitum*). Participants could then be asked to return to the laboratory to consume a snack and complete measures related to memory encoding of the previous meal consumed in the laboratory. A useful tool that could enable accurate estimates of memory encoding would be a computer-based task during which participants are shown a collection of pictures of the food consumed differing slightly in the amount presented. The closer their estimate is to their actual intake, the better the memory encoding would be. It could then be tested whether individuals with low and high ADHD symptoms differ in memory encoding, and if disruptions in this process can predict subsequent increased snack consumption. Another potentially interesting future direction would be to assess whether lisdexamfetamine reduces BED via a direct impact on inattentive symptoms of ADHD. An improved understanding of the specific mechanisms underlying the association between inattentive symptoms of ADHD and binge/disinhibited and overeating behaviour may have

important implications for treatment (behavioural and pharmacological) and findings from the present thesis highlight the importance of the continuation of the present research.

6.5 Conclusions

In summary, the research presented in this thesis, provides novel findings about specific cognitive and neural mechanisms that may serve as risk factors for the development of overeating and disordered eating behaviour, while opening avenues for future research. Our suggested framework of research in ADHD and disordered eating behaviour encompasses different measures of analysis, including self-report measures of disordered eating and laboratory measures of food intake along with performance-based tasks to assess specific cognitive constructs and neural correlates of eating behaviour. In this thesis we provide the first evidence from an experimental study specifically designed to assess eating behaviour in individuals with high ADHD symptomatology. Well-validated laboratory measures of eating behaviour are crucial components of the framework, and the model presented in this thesis can be used for the study of other mental disorders associated with disordered eating behaviour.

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Appendix A: Search Strategy

Search Terms for Each Database

PUBMED

(ADHD OR adhd OR attention deficit disorder with hyperactivity OR minimal brain disorders OR syndrome hyperkinetic OR hyperkinetic syndrome OR hyperactivity disorder OR hyperactive child syndrome OR childhood hyperkinetic syndrome OR attention deficit hyperactivity disorders OR attention deficit hyperactivity disorder OR adhd attention deficit hyperactivity disorder OR addh OR overactive child syndrome OR attention deficit hyperkinetic disorder OR hyperkinetic disorder OR attention deficit disorder hyperactivity OR attention deficit disorders hyperactivity OR child attention deficit disorder OR hyperkinetic syndromes OR syndromes hyperkinetic OR hyperkinetic syndrome childhood) AND (eating disorder* OR feeding disorder* OR EDNOS OR OSFED OR disordered eat* OR body dysmorph* disorder* OR eating behav* OR eating patholog* OR eating psychopatholog* OR abnormal eating OR binge* OR binging OR binge-eating disorder OR grazing OR graze* OR purging OR purge* OR vomiting OR chaotic eating OR bulimia OR bulimia nervosa OR bulimi* behav* OR binge-eating disorder OR anorexia OR anorexia nervosa OR restrictive eating OR restrictive food intake OR selective eating OR avoidant restrictive food intake disorder OR ARFID OR pica OR night eating OR NES OR eating habit* OR eating pattern* OR eating attitude* OR eating problem* OR loss of control OR lack of control OR overeat* OR over eat* OR excessive eat* OR hyperphagia OR compulsive eat* OR compulsive food intake OR excessive appetite).

OVID DATABASES: Medline, PsycINFO, EMBASE+EMBASE CLASSIC

(ADHD OR adhd OR attention deficit disorder with hyperactivity OR minimal brain disorders OR syndrome hyperkinetic OR hyperkinetic syndrome OR hyperactivity disorder OR hyperactive child syndrome OR childhood hyperkinetic syndrome OR attention deficit hyperactivity disorders OR attention deficit hyperactivity disorder OR adhd attention deficit hyperactivity disorder OR addh OR overactive child syndrome OR attention deficit hyperkinetic disorder OR hyperkinetic disorder OR attention deficit disorder hyperactivity OR attention deficit disorders hyperactivity OR child attention deficit disorder OR hyperkinetic syndromes OR syndromes hyperkinetic OR hyperkinetic syndrome childhood OR Attention deficit disorder / OR ((atteni\$) adj3 (deficit\$ OR disorder\$ or hyperactiv\$ OR hyper?activ\$ OR adhd OR addh OR ad??hd)) OR ((hyperkin\$ OR hyper?kin\$) adj3 (deficit\$ OR disorder\$ OR hkd))) AND (eating disorder* OR feeding disorder* OR EDNOS OR OSFED OR disordered eat* OR body dysmorph* disorder* OR eating behav* OR eating patholog* OR eating psychopatholog* OR abnormal eating OR binge* OR bingeing OR binge-eating disorder OR grazing OR graze* OR purging OR purge* OR vomiting OR chaotic eating OR bulimia OR bulimia nervosa OR bulimi* behav* OR binge-eating disorder OR anorexia OR anorexia nervosa OR restrictive eating OR restrictive food intake OR selective eating OR avoidant restrictive food intake disorder OR ARFID OR pica OR night eating OR NES OR eating habit* OR eating pattern* OR eating attitude* OR eating problem* OR loss of control OR lack of control OR overeat* OR over eat* OR excessive eat* OR hyperphagia OR compulsive eat* OR compulsive food intake OR excessive appetite).

ISI WEB of Knowledge

(Web of Science [Science Citation Index Expanded], Biological Abstracts, Biosis, Food Science and Technology Abstracts)

(ADHD OR adhd OR attention deficit disorder with hyperactivity OR minimal brain disorders OR syndrome hyperkinetic OR hyperkinetic syndrome OR hyperactivity disorder OR hyperactive child syndrome OR childhood hyperkinetic syndrome OR attention deficit hyperactivity disorders OR attention deficit hyperactivity disorder OR adhd attention deficit hyperactivity disorder OR addh OR overactive child syndrome OR attention deficit hyperkinetic disorder OR hyperkinetic disorder OR attention deficit disorder hyperactivity OR attention deficit disorders hyperactivity OR child attention deficit disorder OR hyperkinetic syndromes OR syndromes hyperkinetic OR hyperkinetic syndrome childhood) AND (eating disorder OR feeding disorder OR EDNOS OR OSFED OR disordered eating OR body dysmorphic disorders OR eating behavior OR eating pathology OR eating psychopathology OR abnormal eating OR bingeing OR binging OR binge-eating disorder OR grazing OR graze OR purging OR purge OR vomiting OR chaotic eating OR bulimia OR bulimia nervosa OR bulimic behavior OR binge-eating disorder OR anorexia OR anorexia nervosa OR restrictive eating OR restrictive food intake OR selective eating OR avoidant restrictive food intake disorder OR ARFID OR pica OR night eating OR NES OR eating habits OR eating patterns OR eating attitudes OR eating problems OR loss of control OR lack of control OR overeating OR over eating OR excessive eating OR hyperphagia OR compulsive eating OR compulsive food intake OR excessive appetite).

Appendix B: Descriptive Summaries of the Studies

Table 1 Studies presenting data relevant to an association between ADHD and EDs

Reference (Year)/ Country	Type of Study/ Study Name	Participant Characteristics					ADHD Assessment	ED/Eating pathology assessment	Key Results	Confounder s included in analysis (if any)
		Source Population	Sample size (% females)	Case Group (% females)	Control Group (% females)	Age Range (y)/ Mean Age (y) (mean \pm SD)				
Bijlenga et al. (2013) Netherlands	Case-control	ADHD: outpatient clinic for adult ADHD CG: community sample and students	Total N=391 55.8% females	ADHD N=202 47% females	CG N=189 65.1% females	18-65 ADHD: 34.9 \pm 10.6 CG: 33.0 \pm 13.6	DSM-IV criteria; semi-structured diagnostic interview for ADHD in adults	DSM-IV criteria; Use of standard checklist to assess psychiatric comorbidity (including EDs)	In a population of 202 ADHD patients, 5% were comorbid for EDs	-
Bleck & DeBate (2013) US	Secondary data analysis of data from the National Longitudinal Study of Adolescent Health (Wave III)	Nationally representative cohort of adolescents	Total N=4,862 54% females	Nationally representative cohort of adolescents N=4,862	NA	18-28 (21.79 \pm 0.12)	DSM-IV-TR; self-report, participants evaluated their behaviour between 5-12 yrs	Diagnosed EDs: self-report DE behaviours: participants reported any of the following in the past 7 days: 1) in order to lose weight: they fasted or skipped meals, took laxatives, used diuretics, used weight loss pills, made themselves throw up, or took food supplements to take the place of meals or to reduce appetite; or 2) they ate so much in a short period of time that they would be embarrassed if someone saw, or reported being afraid to start eating for fear of losing control	ADHD predicted diagnosed EDs in females (IRR = 2.06; 95% CI: 1.09–3.88; p < .05), but not in males ADHD significantly predicted disordered eating behaviours (OR = 1.82; 95% CI: 1.21–2.74; p < .01)	Age, sex, race
Bleck et al. (2015) US	Secondary data analysis of data from the	Nationally representative cohort of adolescents	Total N=12,262 51% females	Nationally representative cohort of adolescents	NA	18-27 (21.8)	Clinical ADHD; self-report Subclinical ADHD: self-	See Bleck and DeBate (2013) Clinical-level restrictive behaviour: self-report of	Participants with clinical ADHD were 2.81 times more likely to report clinical EDs	Age, sex, race

Table 1 (*continued*)

Reference (Year)/ Country	Type of Study/ Study Name	Participant Characteristics					ADHD Assessment	ED/Eating pathology assessment	Key Results	Confounder s included in analysis (if any)
		Source Population	Sample size (% females)	Case Group (% females)	Control Group (% females)	Age Range (y)/ Mean Age (y) (mean \pm SD)				
Karjalainen et al. (2016) Sweden	Cross- sectional study	Referred adults with ADHD and/or ASD	Total N = 228 44.3% females	Referred adults with ADHD and/or ASD N = 228 44.3% females	NA	19-60 31.75 \pm 9.29	SCID-II	EDs: SCID-I Eating pathology: EAT-26 (sample size limited to 138)	The prevalence of EDs in the ADHD group (n = 109) was 8.3%; higher than the estimated prevalence in general population, suggesting a positive association Only 1% of teenagers SUB for ADHD were comorbid for full symptoms of EDs; 2.9% of teenagers SUB for ADHD were comorbid for SUB EDs (ORs: not reported; not significant) 31.4% of adolescents with EDs, in contrast with the 8.4% of adolescents without EDs	Age, education
Lewinshon et al. (2004) US	Epidemiol ogic study OADP	Adolescent community sample	Total N=1,704 52.1% females	Adolescent community sample N=1,704	NA	16.6 \pm 1.2	Diagnostic interview; K- SADS; SUB diagnoses based on criteria of the DSM-II-R (5 or more symptoms)	Diagnostic interview; K- SADS; SUB diagnoses based on criteria of the DSM-II-R		-
Rojo- Moreno et al. (2015) Spain	Longitudin al study	Community- based sample	Total N = 962 (cross- sectional	Adolescent community sample N = 962	NA	12-16 (cross- sectional analysis) 14-17	K-SADS; semi-structured interview	KSADS correction; specific instructions in order to diagnose AN or BN full-syndrome, partial- syndrome and subclinical ED		-

			analysis) 47.8% females n = 326 (longitudinal analysis) 49.1% females	47.8% females		(longitudinal analysis)			diagnosed with ADHD (Risk = 5.03, 95% CI: 2.37-10.64) In only 2 of the new ED cases the previous psychiatric evaluation was positive	
Sobanski et al. (2007) Germany	Case-control Part of an ongoing study on the genetic basis of adult ADHD	ADHD: clinical; referred patients CG: Population-based	Total N = 140 54.3 % females	ADHD N = 70 54.3% females	CG N = 70 54.3% females	ADHD: 36.8 ± 9.0 CG: 39.8 ± 10.0	DSM-IV; Structured Clinical Interview SCID-I, a semi-structured interview for present and childhood ADHD (Hypescheme), WURS-k, the BADDS and the LPS	DSM-IV; Structured Clinical Interview	The overall lifetime prevalence of EDs estimated to 11.4% in the ADHD compared to 1.4% in the CG ($\chi^2 = 5.930$, df = 1, p = .015) More females than males with ADHD were comorbid for EDs (8.6% vs. 2.9%) ($\chi^2(1) = 4.960$; p = .026)	-
Stulz et al. (2013) Switzerland	Cross-sectional	Females with diagnosed EDs recruited from the ED Unit of the University Hospital of Zurich	Total N=32 100% females	ED patients N=32	NA	17-46 23.1 ± 5.6	ADHD-SR questionnaire	Participants recruited from an ED Unit; SIAB-EX was used to asses specific and general psychopathology of ED	None of the 32 ED patients met criteria for diagnosis of ADHD	-

(continued on next page)

Table 1 (continued)

Reference (Year)/ Country	Type of Study/ Study Name	Participant Characteristics					ADHD Assessment	ED/Eating pathology assessment	Key Results	Confounder s included in analysis (if any)
		Source Population	Sample size (% females)	Case Group (% females)	Control Group (% females)	Age Range (y)/ Mean Age (y) (mean \pm SD)				
Viborg et al. (2014) Sweden	Two-wave longitudinal study with a one-year interval	Community sample of Swedish adolescent girls	Total N = 428 100% females	Community sample of adolescent girls N = 428 100% females	NA	13–15	SDQ-s; Swedish version	RiBED-8	Hyperactivity– inattention was a unique predictor of DE, when DE and BMI at T1 were controlled for (B = 0.29, SE B = 0.10, β = 0.13, $p < .01$).	BMI, disordered eating at baseline
Yoshimasu et al. (2012) US	Cohort Study	Population- based; birth cohort	Total N=1055 25% females	ADHD N = 343 25.1% females	CG N=712 25% females	Follow-up age: 19	Subjects were defined as research- identified ADHD cases if their school/ medical records included various combinations of 3 different categories of information: 1) met DSM-IV criteria for ADHD, 2) positive ADHD questionnaire results, 3) clinical diagnosis of ADHD	Abstraction of medical records In order for an adolescent to be classified with a psychiatric Disorder (including EDs), documentation of (a) the initial diagnosis and (b) separate confirmation of the initial diagnosis was required	Youth with ADHD were nearly 6 times more likely to develop an ED than those without ADHD (HR = 5.68; 95% CI: 1.14–28.21, $p < .005$) by the age of 19 yrs No moderating effect of sex ($p = .67$)	Sex, mother's age and education at the subject's birth

ADHD-SR = Attention Deficit/Hyperactivity Disorder-Self-Rating Screening Questionnaire; AN = Anorexia Nervosa; ASD = Autism Spectrum Disorders; BADDS = Brown Attention Deficit Disorder Scales; BN = Bulimia Nervosa; CG = Control Group; DE = Disordered Eating; DSM-IV = Diagnostic and Statistical Manual of Mental Disorders, 4th Edition; DSM-IV-TR = Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision; ED = Eating Disorder; EDs = Eating Disorders; IRR = Incidence Rate Ratio; K-SADS = Schedule for Affective Disorders and Schizophrenia for School-Age Children; K-SADS-E = Schedule for Affective Disorders and Schizophrenia for School-Age Children–Epidemiologic Version; K-SADS-P = Schedule for Affective Disorders and Schizophrenia for School-Age Children–Present and Lifetime Version; LPS = Leistungsprüfsystem; OADP = Oregon Adolescent Depression Project; OCD = Obsessive-Compulsive Disorder; RiBED-8 = Risk Behaviour related to Eating disorders; SADS-LA = Schedule for Affective Disorders and Schizophrenia–Lifetime Version, Modified for the Study of Anxiety Disorders; SCID-I = The Structured Clinical Interview for Diagnosis, according to the DSM-IV Axis I Disorders; SCID-II = The Structured Clinical Interview for Diagnosis, according to the DSM-IV Axis II Personality Disorders; SCID-I/NP = Structured Clinical Interview for DSM-IV Axis I Disorders–Non-Patient Edition; SDQ-s = The Strengths and Difficulties Questionnaire-self-report version; SIAB-EX = Structured Interview for Anorexia Nervosa and Bulimia Nervosa-Expert Rating; TS = Tourette Syndrome; TSA: Tourette Syndrome Association; US = United States; WURS-k = Wender Utah rating scale

Table 2 Studies presenting data relevant to an association between ADHD and AN/Restrictive eating

Reference (Year)/ Country	Type of Study/ Study Name	Participant Characteristics					ADHD Assessment	ED/Eating pathology assessment	Key Results	Confounders included in analysis (if any)
		Source Population	Sample size (% females)	Case Group (% females)	Control Group (% females)	Age Range (y)/ Mean Age (y) (mean \pm SD)				
Biederman et al. (2007) US	Prospective with 5-year F/UP	ADHD: psychiatric and paediatric clinics CG: selected from list of outpatients at paediatric medical clinics	Total N=235 100% females	ADHD N=123 100% females	CG N=112 100% females	At F/UP: 10 - 25 (ADHD vs. CG: 16.4 vs.17.1)	DSM-III-R defined ADHD from paediatric and psychiatric clinics	Interviews: - K-SADS-E for participants <18 yrs - SCID-NP for participants \geq 18 yrs DSM-IV diagnostic criteria; a subject was classified as having ED if full or SUB DSM criteria were met	Survival results for AN were not statistically significant when ADHD compared to CG (HR = 2.7, 95% CI: 0.89-8.7, p = .09)	-
Biederman et al. (2010) US	Prospective with 11-year F/UP	See Biederman et al. (2007)	Total N=187 100% females	ADHD N=96 100% females	CG N=91 100% females	At F/UP: 15 – 30 (22.1 \pm 3.3)	DSM-III-R- defined ADHD from paediatric and psychiatric clinics	DSM-IV diagnostic criteria; a subject was classified as having ED if full DSM criteria were met	Survival results for AN were not statistically significant when ADHD compared to CG (HR = 2.2, 95% CI: 0.8-5.7, p = 0.11)	Baseline psychopatholo gy (mood, anxiety, antisocial, developmental disorders)
Bleck & DeBate (2013) US	Secondary data analysis of data from the National Longitudinal Study of Adolescent Health (Wave III)	A nationally representativ e cohort of adolescents	Total N=4,862 54% females	Nationally representativ e cohort of adolescents N=4,862	NA	18-28 (21.79 \pm 0.12)	DSM-IV-TR; self-report, participants were asked to evaluate their behaviour between 5-12 yrs old	Disordered eating behaviours (see Table 1)	ADHD did not predict restrictive behaviours (OR = 1.23, 95% CI: 0.75- 2.03)	Age, sex, race

Bleck et al. (2015) US	Secondary data analysis of data from the National Longitudinal Study of Adolescent Health (Wave III-Wave IV)	Nationally representative cohort of adolescents	Total N=12,262 51% females	Nationally representative cohort of adolescents N=12,262 51% females	NA	18-27 (21.8)	Clinical ADHD: self-report Subclinical ADHD: self-report (see Table 1)	Restrictive behaviour: see Bleck and DeBate (2013), Table 1 Clinical-level restrictive behaviour: self-report of clinical ED and current (past week) restrictive behaviours	Participants that reported clinical ADHD were 4.92 times more likely to report clinical level restrictive behaviour (OR=4.92; CI 2.15–11.27; p< .001) Participants that reported subclinical ADHD were not more likely to report restrictive behaviour (p> .05)	Age, sex, race
Blinder et al. (2006) US	Retro-spective chart review	Female ED inpatients	Total N=2,436 100% females	ED N=2,436 520 ANR, 436 ANB; 882 BN; 598 EDNOS	NA	11–68 (23.4 ± 8.6)	WURS; CPRS–R:L and CASS:L Adults admitted after June 1, 1999, completed the SSPQ-X	EDI-II Adults admitted after June 1, 1999, completed the SSPQ-X	ADHD was found in 6% of all ED patients; no significant difference in prevalence according to ED type The prevalence of ADHD in AN patients was 3% (both ANR, ANB) suggesting a non-significant association No difference between OB with and without ADHD symptoms on cognitive restraint (p = .08)	Socio-demographic and severity of-illness measures (e.g., education, marital status, ED onset age, admission BMI)
Dempsey et al. (2011) US	Cross-sectional	OB participants recruited from community sites	Total N=125 65.6% females	OB participants N=125	NA	43.7± 13.47	Current ADHD symptoms: ASRS–VI.1 (6 items)	Cognitive Restraint; EI	-	-

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Table 2 (continued)

Reference (Year)/ Country	Type of Study/ Study Name	Participant Characteristics					ADHD Assessment	ED/Eating pathology assessment	Key Results	Confounders included in analysis (if any)
		Source Population	Sample size (% females)	Case Group (% females)	Control Group (% females)	Age Range (y)/ Mean Age (y) (mean \pm SD)				
Grabarek & Cooper (2008) US	Cross- sectional	Graduate and law students	Total N=67 57% females	Students N=67 57% females	NA	20-25	Inattention and Hyperactivity symptoms of ADHD: BASC-2 Self-Report of Personality– College Level	Drive for thinness: EDI-3 RF; self- report	Hyperactivity-not inattention-was related significantly to Drive for thinness in men ($r = .38$, $p < .05$) but not women ($p > .05$)	-
Hudson et al. (2007) US	Cross- sectional	A representativ e sample of US adults	Total N = 9,282 55.4% females	Total N = 9, 282 55.4% females	NA	>18	Face to face interviews: CIDI version 3.0 based on DSM-IV criteria	Face to face interviews: CIDI version 3.0 based on DSM-IV criteria	AN was not significantly associated with ADHD ($p > .005$)	Age, sex, race/ethnicity
Karjalainen et al. (2016) Sweden	Cross- sectional study	Referred adults with ADHD and/or ASD	Total N = 228 44.3% fem ales	Referred adults with ADHD and/or ASD N = 228 44.3% females	NA	19-60 31.75 \pm 9.29	SCID-II	EDs: SCID-I Eating pathology: EAT-26 (sample size limited to 138)	Individuals with ADHD more often affirmed eating pathology such as focusing on thoughts of calories and body dissatisfaction compared to individuals with ASD EAT variables discriminating significantly between the sub- groups ASD-only	Age, education

									and ADHD-only: 1) Am preoccupied with the thought of having fat on my body (OR = 0.49, 95% CI: 0.26–0.92, p = .0259) 2) Enjoy trying new rich foods (OR = 1.60, 95% CI: 1.02–2.53, p = .0428)	
Malmberg et al. (2011)	Part of Longitudinal study	A population-based sample of twins	Total N=312	Population-based sample;	NA	14.6- 16.7 (mean=16)	SUB diagnoses based on criteria of the DSM-IV TR; Swedish K-SADS-PL version 15; structured interview with one child's parent & separate interview with the child	AN symptoms; "Fear of becoming obese" item; K-SADS-PL screening interview	SUB diagnoses of ADHD-comb was positively associated with symptoms of AN (item fear of becoming obese of the K-SADS-PL screening interview) (OR: 3.29, 95% CI: 1.04-10.41)	-
Sweden	The TCHAD-study		57% females	N=312	57% females					
Muller et al. (2014)	Cross-sectional	Bariatric surgery candidates and OB inpatients from a psychosomatic unit	Total N=156	Bariatric surgery candidates and OB inpatients;	NA	18–65 (39.91±11.42)	Childhood ADHD symptoms: WURS-k	Restraint: EDE-Q	Analysis revealed a (1)'resilient/high functioning' cluster (n = 88) & an (2)'emotionally dysregulated/under-controlled' cluster (n = 68)	-
Germany			72% females	Total N=156			Adult ADHD symptoms: ADHD-SR; DSM-IV criteria		Cluster 2 showed more ED (p< .001), depressive (p = .004) and ADHD symptoms (p< .0001) but did not score higher on the restraint (p= .080)	

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Table 2 (*continued*)

Reference (Year)/ Country	Type of Study/ Study Name	Participant Characteristics					ADHD Assessment	ED/Eating pathology assessment	Key Results	Confounders included in analysis (if any)
		Source Population	Sample size (% females)	Case Group (% females)	Control Group (% females)	Age Range (y)/ Mean Age (y) (mean \pm SD)				
Pauli-Pott et al. (2013) Germany	Cross- sectional	OW/ OB children who had been referred for weight reduction	Total N=128 48% females	ADHD; clinical symptoms- N=17, subclinical symptoms N=71	CG; no ADHD symptoms N=40	7.5-15 (11.0 \pm 1.81)	ADHD Symptoms: K-SADS- PL, Version 1.0; 25- screen interview	Restraint Eating; EPI	ADHD groups did not differ in dietary restraint ($p > .05$) No significant interaction effects with sex	Sex, age, ODD/CD symptoms
Rastam et al. (2013) Sweden	Ongoing longitudinal study The CATSS study	All twins Born in Sweden since July 1, 1992	Total N=12,366 48.5% females	All twins born in Sweden since July 1, 1992; N=12.366	NA	9 and 12	The A-TAC Inventory, lower cut-offs for screening positive for ADHD; parental telephone interviews	Eating Module for restrictive eating problems (A-TAC inventory) (2 questions); parental telephone interviews	Only 3 variables associated with restrictive eating: Social interaction problems for girls (OR = 1.95, 95% CI: 1.22-3.10; $p < .005$) & for boys Impulsiveness/Acti vity problems (OR = 1.41, 95% CI: 1.18-1.69; $p < .001$), and age 9 years compared to 12 years (OR = 0.37, 95% CI: 0.16-0.84; $p < .05$) Female offenders with ADHD were 6.2 times more likely to suffer from AN than non- ADHD controls (OR = 6.2, 95% CI:	-
Rosler et al. (2009) Germany	Cross- sectional	Female offenders	Total N=94 100% females	ADHD Group; N=11	N=83	34 \pm 12	WRI; Retrospective assessment of childhood ADHD (WURS-k); diagnosis of ADHD in	SCID-I interview according to DSM-IV	Female offenders with ADHD were 6.2 times more likely to suffer from AN than non- ADHD controls (OR = 6.2, 95% CI:	-

							accordance to DSM-IV or ICD-10 research version (ADHD-DC)		1.5–26.5)	
Slane et al. (2010)	Cross-sectional	Twins recruited for the MSUTR	Total N=541	Twins recruited for the MSUTR;	NA	20.89 ± 2.33	Only hyperactivity assessed; Hyperactivity subscale of the Current Symptoms Scale-Self-Report Form	Dietary Restraint: Restraint subscale of the EDEQ	There was an interaction between sex and hyperactivity (hyper); the association between hyper and dietary restraint was significantly stronger in men than women (Model for Restraint, Pseudo R ² = 0.29, B= 0.09, p = .04)	BMI
US	Michigan State University Twin Registry (MSUTR)		62% females	N=541						
Swanson et al. (2011)	Cross-sectional	A representative sample of US adolescents	Total N = 10,123	A representative sample of US adolescents	NA	13-18	Parent self-report questionnaire (SAQ) based on DSM-IV criteria	Face to face interviews: Modified version of CIDI to assess AN, BN, BED based on DSM-IV criteria; SUB EDs assessed using diagnostic algorithms	AN and SAN was not significantly associated with ADHD (p> .005)	Age, sex, race/ethnicity
US			48.7% females	N = 10 123 48.7% females				DSM-IV ED criteria; all DSM IV ED diagnoses were retrospectively re-categorized according to the DSM-5 criteria		
Welch et al. (2015)	Retrospective review	Referred patients for medical assessment of diagnosed or suspected ED	Total N=664	ED patients; N=664 (Boys were diagnosed with either AN or atypical anorexia)	NA	<18 years; 14.9 ± 1.6 for males; 15.2 ± 1.7 for females	No direct assessment; previously established psychiatric diagnoses were registered		6.9% of males vs. 1.0% of females had a diagnosis of ADHD, suggesting a significant association between ADHD and AN, atypical anorexia in males	
Sweden			91% females							

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Table 2 (*continued*)

Reference (Year)/ Country	Type of Study/ Study Name	Participant Characteristics					ADHD Assessment	ED/Eating pathology assessment	Key Results	Confounders included in analysis (if any)
		Source Population	Sample size (% females)	Case Group (% females)	Control Group (% females)	Age Range (y)/ Mean Age (y) (mean \pm SD)				
Wentz et al. (2005) UK	Cross- sectional	Clinical sample with ED; inpatients and outpatients	Total N=30 100% females	ED N=30 21 AN, 9 BN	NA	18- 56 (27.4 \pm 8.4)	ADHD-RS interview; DSM- IV criteria	AN or BN; Diagnostic criteria according to DSM-IV	3 ED patients met criteria for childhood-onset of ADHD; all were ANB cases	-
Yates et al. (2009) US	Cross- sectional data; study was part of a longitudinal follow-up study (LOGI)	ED inpatients	Total N=189 100% females	ED inpatients N=189; 152 AN, 37 BN	NA	65% of study participants were < 18 yrs	Childhood and current ADHD symptoms: MINI	SCID-I; SIAB	The overall rate for a full diagnoses of ADHD was 5.8% (95% CI: 2.6%– 9.5%), suggesting a non-significant association between ADHD and ED, mainly AN Current inattention symptoms differed significantly by ED (F =13.2, df = 2, p< .001); higher inattention total symptoms for BN compared with ANB and ANR subtype	-

ADHD-comb = ADHD combined type; ADHD-DC = ADHD Diagnostic Checklist; ADHD-RS = ADHD Rating Scale; ADHD-SR = ADHD Self-Rating scale; AN = Anorexia Nervosa; ANB = Anorexia Nervosa Binge-Eating/Purging type; ANR = Anorexia Nervosa Restrictive type; ASD = Autism Spectrum Disorders; ASRS = The World Health Organization Adult AD/HD Self-Report Scale Adult Self Rating Scale; A-TAC = The Autism-Tics, ADHD, and other Comorbidities Inventory; BASC = Behavior Assessment System for Children; BED = Binge Eating Disorder; BN = Bulimia Nervosa; CASS-L Conners-Wells Adolescent Self-Report Scale (Long Version); CATSS = Child and Adolescent Twin Study in Sweden; CG = Control Group; CIDI = Composite International Diagnostic Interview; CPRS-R:L = Conners' Parent Rating Scale-Revised (Long Version); DSM-III-R = Diagnostic and Statistical Manual of Mental Disorders, 3rd Edition Revised Version; DSM-IV = Diagnostic and Statistical Manual of Mental Disorders, 4th Edition; DSM-IV-TR = Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision; EAT = Eating Attitudes Test; EDE-Q = Eating Disorder Examination-Questionnaire ; EDI-II = Eating Disorders Inventory, 2nd Edition; EDI-3 RF = Eating Disorder Inventory-Referral Form; EI = Eating Inventory; EDNOS = Eating Disorder Not Otherwise Specified; EPI = Eating Pattern Inventory; F/UP = Follow-up; K-SADS-E = Schedule for Affective Disorders and Schizophrenia for School-Age Children-Epidemiologic Version; K-SADS-PL = Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version; MINI = Multi international Psychiatric Interview ; OB = Obese; OW = Overweight; SAN = Subthreshold Anorexia Nervosa; SAQ = Self-Administered Questionnaire; SCID-I = The Structured Clinical Interview for Diagnosis, according to the DSM-IV Axis I Disorders ; SCID-II = The Structured Clinical Interview for Diagnosis, according to the DSM-IV Axis II Personality Disorders ; SCID-NP = Structured Clinical Interview for DSM-III-R: Non-Patient Edition; SIAB = The Structured Interview for Anorexia Nervosa and Bulimia Nervosa; SSPQ-X = SCID Screen Patient Questionnaire-Extended; SUB = Subthreshold; TCHAD = Swedish Twin study of Child and Adolescent Development; WRI = The Wender-Reimherr interview ; *Wurs*: *Wender Utah Rating Scale*; Wurs-k = Wender Utah rating scale, German short version

Table 3 Studies presenting data relevant to an association between ADHD and BN

Participant Characteristics										
Reference (Year)/ Country	Type of Study/ Study Name	Source Population	Sample size (% females)	Case Group (% females)	Control Group (% females)	Age Range (y)/ Mean Age (y) (mean ± SD)	ADHD Assessment	ED/Eating pathology assessment	Key Results	Confounders included in analysis (if any)
Biederman et al. (2007) US	Prospective with 5-year F/UP	ADHD: psychiatric and paediatric clinics CG: selected from list of outpatients at paediatric medical clinics	Total N=235 100% females	ADHD N=123 100% females	CG N=112 100% females	At F/UP: 10 - 25 (ADHD vs. Control: 16.4 vs.17.1)	DSM-III-R defined ADHD from paediatric and psychiatric clinics	Interviews: - K-SADS-E for participants <18 yrs - SCID-NP for participants >=18 yrs DSM-IV diagnostic criteria; a subject was classified as having BN if full or SUB DSM criteria were met	ADHD females were 5.6 times more likely to meet criteria for BN compared to controls (HR = 5.6; 95% CI: 1.6 – 19.0, p< .01) throughout the F/UP	-
Biederman et al. (2010) US	Prospective with 11-year F/UP	See Biederman et al. (2007)	Total N=187 100% females	ADHD N=96 100% females	CG N=91 100% females	At F/UP: 15 – 30 (22.1 ± 3.3)	DSM-III-R- defined ADHD from paediatric and psychiatric clinics	DSM-IV diagnostic criteria; a subject was classified as having BN if full DSM criteria were met	ADHD females were 5.2 times more likely to meet criteria for BN compared to controls (HR = 5.2; 95% CI: 2.0 – 13.7, p = .001) throughout the F/UP	Baseline psychopatholo gy (mood, anxiety, antisocial, developmental , disorders)

Bleck & DeBate (2013)	Secondary data analysis of data from the National Longitudinal Study of Adolescent Health (Wave III)	A nationally representative cohort of adolescents	Total N=4,862 54% females	Nationally representative cohort of adolescents N=4,862	NA	18-28 (21.79± 0.12)	DSM-IV-TR; self-report, participants were asked to evaluate their behaviour between 5-12 yrs old	Disordered eating behaviours (see Table 1)	ADHD significantly predicted bingeing and/or purging behaviour (OR = 2.86, 95% CI: 1.78–4.61; p< .001)	Age, sex, race
Bleck et al. (2015)	Secondary data analysis of data from the National Longitudinal Study of Adolescent Health (Wave III-Wave IV)	Nationally representative cohort of adolescents	Total N=12,262 51% females	Nationally representative cohort of adolescents N=12,262 51% females	NA	18-27 (21.8)	Clinical ADHD: self-report Subclinical ADHD: self-report (see Table 1)	Bingeing and/or purging behaviour: see Bleck and DeBate (2013), Table 1 Clinical-level bingeing and/or purging behaviour: self-report of clinical ED and current (past week) bingeing and/or purging behaviours EDI-II	Participants with clinical ADHD were 8.14 times more likely to report clinical-level bingeing and/or purging (OR = 8.14, 95% CI 3.50–18.95; p< .001). Participants with subclinical ADHD were more likely to report bingeing and/or purging (p> .05)	Age, sex, race
Blinder et al. (2006)	Retrospective chart review	Female ED inpatients	Total N=2,436 100% females	ED N=2,436 520 ANR, 436 ANB; 882 BN 598 EDNOS	NA	11–68 (23.4 ± 8.6)	WURS; CPRS-R:L and CASS:L Adults admitted after June 1, 1999 completed the SSPQ-X	Adults admitted after June 1, 1999, completed the SSPQ-X	ADHD was found in 6% of all ED patients; no significant difference in prevalence according to ED type The prevalence of ADHD in BN patients was 9%, suggesting a positive association	Socio-demographic and severity of-illness measures (e.g. education, marital status, ED onset age, admission BMI)

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Table 3 (continued)

Reference (Year)/ Country	Type of Study/ Study Name	Participant Characteristics					ADHD Assessment	ED/Eating pathology assessment	Key Results	Confounder included in analysis (if any)
		Source Population	Sample size (% females)	Case Group (% females)	Control Group (% females)	Age Range (y)/ Mean Age (y) (mean \pm SD)				
Cortese et al. (2007) France	Cross-sectional	Severely OB adolescents participating in weight-loss programme	Total N=99 66% females	OB adolescents N=99	NA	12–17 (14.2 \pm 1.2)	CPRS	BITE; self-report	Bulimic behaviours were significantly associated with CPRS-ADHD index scores after controlling for depressive and anxiety symptoms (standardized beta = 0.246, p = .043), but not with hyperactive – impulsive scores (p > .05)	Depressive, anxiety symptoms
De Zwaan et al. (2011) Germany	Cross-sectional	A representative sample of the German general population	Total N=1,633 53.6% females	ADHD N=77	CG N=1,556 Age-and sex matched	43.2 \pm 2.7	ADHD childhood symptoms: retrospectively; WURS-k Adult ADHD symptoms: ADHD-SR	OBEs and purging behaviors during the past 4 weeks: DSM-IV-TR criteria; EDE-Q, (German version)	13% of ADHD individuals reported OBEs compared with 3.5% of controls (χ^2 = 17.646, df = 1, p < .001) 7.8% of ADHD individuals reported purging behaviour compared to 2.6% of controls (χ^2 = 7.307, df = 1, p < .01)	-

Grabarek & Cooper (2008) US	Cross-sectional	Graduate and law students	Total N=67 57% females	Students N=67	NA	20-25	Inattention and Hyperactivity symptoms of ADHD: BASC-2 Self-Report of Personality–College Level	Bulimia; EDI-3 RF; self-report	No significant association between hyperactivity-inattention symptoms of ADHD and bulimia symptoms was found ($r = -0.015$; $p > .05$)	-
Hinshaw et al. (2012) US	Prospective with 10-year F/UP	Both ADHD and controls were participants in summer research camps	Total N=228 (Retention rate, 95%) 100% females	ADHD N=140	N= 88 Age and ethnicity matched	At F/UP: 17–24 (19.6)	Caregivers and young adults completed rating scales (SNAP-IV) and interviews (DISC-IV-YA)	EDI-II; Bulimia subscale, self-report	Participants with childhood-diagnosed ADHD did not differ significantly in terms of eating pathology compared to control group ($F(8,384) = 0.90$, $p = .519$)	Participant age, family income, maternal education, IQ comorbidities for the girls with ADHD (ODD or CD depression/diagnostic thymic disorder or anxiety disorder, reading disorder), psychotropic medication use during the year before the 10-year F/UP Age, sex, race/ethnicity
Hudson et al. (2007) US	Cross-sectional	A representative sample of US adults	Total N = 9,282 55.4% females	Total N = 9,282 55.4% females	NA	>18	Face to face interviews: CIDI version 3.0 based on DSM-IV criteria	Face to face interviews: CIDI version 3.0 based on DSM-IV criteria	BN was significantly associated with ADHD (OR = 8.4; 95% CI=3.1-22.6; $p < .05$)	

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Table 3 (*continued*)

Reference (Year)/ Country	Type of Study/ Study Name	Participant Characteristics					ADHD Assessment	ED/Eating pathology assessment	Key Results	Confounder included in analysis (if any)
		Source Population	Sample size (% females)	Case Group (% females)	Control Group (% females)	Age Range (y)/ Mean Age (y) (mean \pm SD)				
Mikami et al. (2008) US	Prospective with 5-year F/UP	Both ADHD and controls were participants in summer research camps	Total N=209 100% females	ADHD N=127 100% females	CG N=82 100% females	At F/UP: 11–18 (14.2)	Clinical cut-offs on parent and teacher rating scales (CBCL; TRF; SNAP-IV) DSM-IV diagnosis was validated in parent clinical interview	At F/UP parents reported on DISC- IV, to yield symptom counts and diagnosis of BN	No girl met diagnostic criteria for BN Childhood ADHD diagnosis was related to the parent-reported BN symptom count (F (2,134) = 5.03; $p < .01$) Impulsivity symptoms predicted Desire to Lose Weight (r = 0.17; $p < .05$), and Pathological Eating (r = 0.13; $p < .05$), in partial correlations controlling for both inattention and hyperactivity symptoms Inattention or hyperactivity never predicted eating pathology with control of impulsivity	Age, BMI, IC baseline satisfaction with physical appearance, baseline disruptive an internalizing comorbidity, early puberty and stimulan medication use

Mikami et al. (2010)	Prospective with 8-year F/UP	Both ADHD and controls were participants in the MTA study	Total N=696 21.0% females	ADHD N=432	CG N=264 Age, sex and ethnicity matched	At F/UP: 15-18 (16.3)	SNAP-IV; parental report	EDI-II; Adolescents self-report DISC-IV; parents and youths self-reports For youths>17 only youth self-report (DIS-IV)	No girl met diagnostic criteria for BN Youth with ADHD displayed more BN symptoms than controls (F (10,448) = 3.38; p < .001) After statistical control of ADHD diagnosis and sex, only impulsivity, and not inattention or hyperactivity, predicted BN symptoms The positive relationship (impulsivity-parent-reported BN symptoms) was stronger for girls (b = 0.49; p < .001) than for boys (b = 0.18; p < .001); (z = 3.79; p< .001) The ADHD group showed a significantly higher score when compared with the non-ADHD group in BITE (p<0.01)	Ethnicity, family income, parent-reported BN symptoms at the 24-month as well as age, pubertal, development and BMI at the 8-year follow-up
Nazar et al. (2012)	Cross-sectional	Women seeking nonsurgical treatment of obesity	Total N=106 100% females	ADHD N=30 100% females	CG N=76 100% females	38.99±10.74	K-SADS module for ADHD, adapted for adults	BITE	-	

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Table 3 (continued)

Reference (Year)/ Country	Type of Study/ Study Name	Participant Characteristics					ADHD Assessment	ED/Eating pathology assessment	Key Results	Confounder included in analysis (if any)
		Source Population	Sample size (% females)	Case Group (% females)	Control Group (% females)	Age Range (y)/ Mean Age (y) (mean \pm SD)				
Neumark – Sztainer et al. (1995) US	Cross- sectional Minnesota Adolescent Health Survey	Population based data	Total N= 3,497 52.6% females	ADHD N=689 58% females	CG N=1,371 adolescents without any chronic illness 45.5% females	12-20 (14.9)	Chronic illness was based on self-report; participants were asked if they had ADHD requiring medication	Participants answered single items about body satisfaction and eating practices including binge eating, dieting, vomiting and use of laxatives, diuretics or emetics to control body weight	All participants with ADHD were more likely to binge, diet and vomit than controls Girls, but not boys, with ADHD were more likely to use laxatives/ diuretics/emetics than controls (OR: 3.74, 95% CI: 1.68-8.32)	Age, race, BMI, and SE
Rosler et al. (2009) Germany	Cross- sectional	Female offenders	Total N=94 100% females	ADHD N=11	CG N=83	34 \pm 12	WRI; Retrospective assessment of childhood ADHD (WURS- k); diagnosis of ADHD in accordance to DSM-IV or ICD-10 research version (ADHD-DC)	SCID-I interview according to DSM- IV	Female offenders with ADHD were 13 times more likely to suffer from BN (OR= 13.0, 95% CI: 3.2-53.7) than non-ADHD controls	-
Seitz et al. (2013) Germany	Case- Control	Women searching treatment for BN; CG recruited via adverts	Total N= 97 100% females	BN N=57	CG N= 40 Age, IQ and BMI matched	15-35 (BN vs. CG; 20.8 \pm 4.82 vs. 21.2 \pm 3.99	1 ADHD interview & 2 ADHD questionnaires (WRI, ADHD- SB & WURS-k)	EDI-II & SIAB-EX was administered to confirm diagnoses and test ED severity	10.7% of females with BN met the full criteria for ADHD vs. 2.5% of controls (OR: 4.2, 95% CI: not reported)	-

Surman et al. (2006) US	4 Case-Control studies	Paediatric studies: Paediatric and psychiatric sources	Paediatric studies (n=2): Total N=522	ADHD Paediatric studies (n=2): N=280	CG Paediatric studies (n=2): N=242	Paediatric studies (n=2): 6-17	DSM-III-R-based structured interviews	DSM-III-R diagnostic criteria; Interviews: - K-SADS-E for participants <18 yrs - SCID-NP for participants ≥18 yrs supplemented with modules from K-SADS-E covering childhood diagnosis	Inattention explained more of the variance of ED symptoms than either impulsivity or hyperactivity (linear regression, $F = 7.50$, $p < .001$; $\beta_{\text{inattention}} = 0.323$, $p = 0.001$; $\beta_{\text{hyperactivity}} = 0.199$, $p = 0.261$; and $\beta_{\text{impulsivity}} = 0.170$, $p = 0.330$) Paediatric samples: The prevalence of BN did not differ between boys and girls with and without ADHD; only 1% of ADHD girls reported BN Adult samples: Greater rates of BN were identified in women with vs. without ADHD (12% vs. 3 %, $p < .05$ for 1 sample and 11% vs. 1%, $p < .05$ for the other sample). No differences were found in men; rates of BN were negligible	-
		Adult studies: Referred adults with ADHD; CG-non-referred adult parents of non-ADHD children (paediatric studies)	50% females Adult Studies (n=2): Total N=742 42% females	Adult Studies (n=2): N=320	Adult Studies (n=2): N= 422	Adult Studies (n=2): Mean age range: 36.9 – 41.3		Diagnosis of children <12 yrs were based on independent interviews with the mothers		

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Table 3 (continued)

Reference (Year)/ Country	Type of Study/ Study Name	Participant Characteristics					ADHD Assessment	ED/Eating pathology assessment	Key Results	Confounder included in analysis (if any)
		Source Population	Sample size (% females)	Case Group (% females)	Control Group (% females)	Age Range (y)/ Mean Age (y) (mean ± SD)				
Swanson et al. (2011) US	Cross-sectional	A representative sample of US adolescents	Total N = 10,123 48.7% females	A representative sample of US adolescents N = 10,123 48.7% females	NA	13-18	Parent self-report questionnaire (SAQ) based on DSM-IV criteria	Face to face interviews: Modified version of CIDI to assess AN, BN, BED based on DSM-IV criteria	BN was significantly associated with ADHD (OR = 3.6, 95% CI=1.0-12.6; p< .05)	Age, sex, race/ethnicity

ADHD-DC = ADHD Diagnostic Checklist; ADHS-SB =ADHD Self-Rating Scale; ADHD-SR = ADHD Self-Rating Scale; BASC = Behavior Assessment System for Children; BES = Binge Eating Scale; BITE = Bulimic Investigatory Test, Edinburg; BN = Bul Nervosa; CBCL = Child Behavior Checklist; CD = Conduct Disorder; CASS:L = Conners-Wells Adolescent Self-Report Scale (Long Version); CG = Control Group; CIDI = Composite International Diagnostic Interview; CPRS: Conner's Parent Rating Scale; CPRS = Conners' Parent Rating Scale-Revised (Long Version); DIS-IV = Diagnostic Interview Schedule for the DSM-IV; DISC-IV: Diagnostic Interview Schedule for Children-4th Edition; DISC-IV-YA = Diagnostic Interview Schedule for Children-4th Edition, Young A version; DSM-III-R = Diagnostic and Statistical Manual of Mental Disorders, 3rd Edition Revised Version; DSM-IV-TR= Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision; ED = Eating Disorder; EDI-II = Eating Disorders Inventory Edition; EDI-3 RF = Eating Disorder Inventory-Referral Form; EDE-Q = The Eating Disorder Examination Questionnaire; F/UP = Follow-up; K-SADS = Schedule for Affective Disorders and Schizophrenia for School-Age Children; K-SADS-E = Schedule for Affective Disorders and Schizophrenia for School-Age Children-Epidemiologic Version; MTA= Multi-Modal Treatment Study of Children with ADHD; OB = Obese; OBEs = Objective Binge Eating Episodes; ODD: oppositional defiant disorder; SAQ = Self-Administ Questionnaire; SCID-NP = Structured Clinical Interview for DSM-III-R: Non-Patient Edition; SCID-I = The Structured Clinical Interview for Diagnosis, according to the DSM-IV Axis I Disorders; SIAB-EX = Structured Interview for Anorexia and Bulimia; SNAP-Swanson, Nolan, and Pelham Rating Scale-4th Edition; SSPQ-X = SCID Screen Patient Questionnaire-Extended; TRF = Teacher Report Form; US = United States; WRI = The Wender-Reimherr interview ; *WURS* = *Wender Utah Rating Scale*; WURS-k = Wender Rating Scale, German short version

Table 4 Studies presenting data relevant to an association between ADHD and BED

Reference (Year)/ Country	Type of Study/ Study Name	Participant Characteristics					ADHD Assessment	ED/Eating pathology assessment	Key Results	Confounder included in analysis (if any)
		Source Population	Sample size (% females)	Case Group (% females)	Control Group (% females)	Age Range (y)/ Mean Age (y) (mean \pm SD)				
Alfonsson et al. (2012) Sweden	Cross- sectional	OB patients presenting for bariatric surgery	Total N=187 73.3% females	Candidates for bariatric surgery N=187	NA	41.04 \pm 07	Adult ADHD: ASRS (18 items)	EDO	The prevalence of BED was not higher in the participants who screened positive for adult ADHD vs. those who screened negative (numbers not presented)	-
Agranat-Meged et al. (2005) Israel	Cross- sectional	OB hospitalised children	Total N= 26 50% females	OB hospitalised children N=26	NA	8–17 years (13.04 \pm 2.78)	The checklist provided in the DSM-IV; the K- SADS-PL and the Conners questionnaire	Both child and caretaker Interviews; DSM-IV ED criteria	57.7% of children were comorbid for ADHD; none of the children met the criteria for BED	-
Bleck & DeBate (2013) US	Secondary data analysis of data from the National Longitudinal Study of Adolescent Health (Wave III)	A nationally representativ e cohort of adolescents	Total N=4,862 54% females	Nationally representativ e cohort of adolescents N=4,862	NA	18-28 (21.79 \pm 0.12)	DSM-IV-TR; self-report, participants were asked to evaluate their behaviour between 5-12 yrs old	Disordered eating behaviours (see Table 1)	ADHD significantly predicted binging and/or purging behaviour (OR = 2.86; 95% CI: 1.78–4.61; p< .001)	Age, sex, rac
Bleck et al. (2015) US	Secondary data analysis of data from the National Longitudinal Study of Adolescent	Nationally representativ e cohort of adolescents	Total N=12,26 2 51% females	Nationally representativ e cohort of adolescents N=12,262 51% females	NA	18-27 (21.8)	Clinical ADHD: self-report; Subclinical ADHD: self- report (see Table 1)	Binging and/or purging behaviour: see Bleck and DeBate (2013), Table1 Clinical-level	Participants with clinical ADHD were 8.14 times more likely to report clinical- level bingeing and/or purging	Age, sex, race

Health (Wave III- Wave IV)							that they had ADHD)	binging and/or purging behaviour: self-report of clinical ED and current (past week) binging and/or purging behaviours	behaviour (OR = 8.14; 95% CI 3.50–18.95; p< .001). Participants that reported subclinical ADHD were more likely to report binging and/or purging behaviour (p< .05). Symptoms of ADHD were significantly, but not differentially elevated in OB with and without BE compared to NW individuals	-
Davis et al. (2009)	A case double control study	Community sample; OB with BE, OB without BE, normal- weight individuals	Total N=181 80.6% females	OB BE N=60	OB non-BE: N=60 NW non-BE: N=61	25-45 (OB BE: 34.5± 6.5, NW: 33.3 ± 7.5; OB non- BE: 37.0 ± 6.7)	Childhood ADHD: WURS Adults ADHD symptoms: CAARS-S-SV	BE: participants had to report at least weekly OBEs over the previous 3 months, but over this period they must not have vomited, fasted, or taken laxatives or diuretics as a means of controlling their shape or weight		
Canada								Food bingeing: Shorter PROMIS Questionnaire	The high-ADHD symptom group had more elevated scores than either the low-ADHD symptom (p = 0.001) or the ADHD group (p = 0.027), who did not differ from each other. The interaction term ADHD x sex was not significant	
Davis et al. (2015)	Cross- sectional	Community- based sample	Total N = 213 53.9% females	ADHD N=46 45.7% females	High-ADHD symptom: N= 83 63.9% females Low-ADHD symptom: N=84 48.8% females	17-32	ADHD Diagnosis: self- report Adult ADHD symptoms: CAARS-S-SV			
Canada										

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Table 4 (*continued*)

Participant Characteristics										
Reference (Year)/ Country	Type of Study/ Study Name	Source Population	Sample size (% females)	Case Group (% females)	Control Group (% females)	Age Range (y)/ Mean Age (y) (mean \pm SD)	ADHD Assessment	ED/Eating pathology assessment	Key Results	Confounder included in analysis (if any)
De Zwaan et al. (2011) Germany	Cross-sectional	A representative sample of the German general population	Total N=1,633 53.6% females	ADHD N=77	CG N=1,556 Age-and sex matched	43.2 \pm 2.7	ADHD childhood symptoms: retrospectively; WURS-k Adult ADHD symptoms: ADHD-SR ASRS-VI.1	OBEs and purging behaviors during the past 4 weeks: DSM-IV-TR criteria; EDE-Q (German version)	13% of ADHD individuals reported OBEs vs. 3.5% of controls ($\chi^2 = 17.646$, df = 1, $p < .001$)	-
Docet et al. (2012) Spain	Case-control	OB patients attending the Nutrition Section of the University Hospital of Vigo	Total N= 230 80.4% females	ADHD N=51 88.2% females	CG N=179 78.2% females	18-79 ADHD: 42.3 \pm 15.5 CG: 40.6 \pm 5.9		Clinical interview; eating pattern questionnaire used in daily clinical practise	OB patients with ADHD were almost 4 times more likely to go on binge eating episodes than the CG (54.9% vs. 23.5%, $\chi^2 = 18.529$; $p = .00$; OR=3.97)	-
Goldschmidt et al. (2015) US	Longitudinal study Pittsburgh Girls Study	Community-based sample	Total N = 2,450 100% females	Community-based sample N= 2,450 100% females	NA	Adolescents: 16 yrs	Hyperactive-Impulsive Scale of CSI-4; parental report at age 10	ChEAT; binge eating was ascertained by asking the respondent how often, in the past year (ranging from 1, “never,” to 6, “always”), she has “gone on eating binges where [she] feel[s] that [she]	Binge eating at age 12 mediated the relation between impulsivity at age 10, and changes in BMI from age 10 to 16 (indirect effect estimate = 0.0007; 95% CI: 0.0001–0.0020).	Age 10 BMI, race/ethnicity SES, verbal comprehension, and binge-eating tendencies

								might not be able to stop”	Binge-eating tendencies at age 14 were not a significant mediator (p> .05)	
Gruss et al. (2012)	Cross-sectional	OB patients considering bariatric surgery	Total N=116 73.3% females	OB patients considering bariatric surgery N=116	NA	38.6 ± 10.5	Childhood ADHD symptoms: WURS-k Adult ADHD symptoms: ADHD-SR	Charts of the clinical evaluation; DSM-IV criteria	The prevalence of BED did not differ between individuals who met childhood-onset ADHD diagnosis and those who were not (35.7% vs. 35.3% respectively)	-
Germany										
Hudson et al. (2007)	Cross-sectional	A representative sample of US adults	Total N = 9,282 55.4% females	Total N = 9,282 55.4% females	NA	>18	Face to face interviews: CIDI version 3.0 based on DSM-IV criteria	Face to face interviews: CIDI version 3.0 based on DSM-IV criteria; BED diagnosis required regular eating binges for 3 (instead of 6) months; SBED assessed (did not require DSM-IV criterion B or C; “any binge eating” also assessed (including BN, BED, SBED, cases of AN with binge eating)	BED was significantly associated with ADHD (OR = 3.1, 95% CI: 1.5-6.2; p< .05); SBED was not significantly associated with ADHD (p> .05); Any Binge Eating was significantly associated with ADHD (OR = 3.0, 95% CI=1.7-5.4; p< .05)	Age, sex, race/ethnicity
US										

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Table 4 (*continued*)

Reference (Year)/ Country	Type of Study/ Study Name	Participant Characteristics					ADHD Assessment	ED/Eating pathology assessment	Key Results	Confounder included in analysis (if any)
		Source Population	Sample size (% females)	Case Group (% females)	Control Group (% females)	Age Range (y)/ Mean Age (y) (mean \pm SD)				
Khalife et al. (2014) Finland	Cohort study NFBC 1986	Population based sample	Total N= 8,106 (at 8 yrs); 49.1% females N= 6, 934 (at 16 yrs); 51.9 % females		NA	Adolescents: 16 years	ADHD assessment: Teachers assessed child behavior of 8- year- olds using the Rutter B2 scale	Binge eating: “How often do you devour large amounts of food?” Binge eating was dichotomized into yes (once a month/once a week/2 or 3 times a week/daily) vs. no (never/hardly ever/ occasionally)	Childhood ADHD symptoms were not found to be linked with binge eating in adolescence (16 yrs) ($p > .05$)	Sex, family structure change (from 8 to 16 yrs), and maternal education
Levy et al. (2009) Canada	Longitudinal clinical intervention study (relevant data cross- sectional)	Referred severely OB adults for the treatment of refractory obesity	Total N=78 92.3% females	Referred severely OB adults with ADHD N=78 92.3% females	NA	41.3	Standard screening tests identified subjects likely to have ADHD. Diagnosis confirmed by interviews; diagnostic (n=16) & semi- structured clinical interviews (n=62)	Symptoms establishing the diagnosis of binge eating disorder were elicited by the examining physician	65.4% (n=51) of the severely OB ADHD individuals were comorbid for BED	
Mattos et al. (2004) Brazil	Cross- sectional	Adult ADHD clinical sample	Total N=86 47% females	Adult ADHD clinical sample Total	NA	18 -52 (ADHD with ED: 28.6 \pm 7.0; ADHD without	ADHD symptoms were investigated either through the patient’s own	SCID-R by trained psychiatrists and psychologists	9 (10.4%) of 86 ADHD patients had an ED, with present BED being the most common one (7	-

				N=86		ED:32.7 ± 9.4)	recollections or parent's report, using the ADHD Childhood Symptoms Scale; DSM-IV criteria		cases). Women were twice more common in the ADHD-ED group, but this difference was not significant (p = .3)
Muller et al. (2012)	Cross-sectional	Morbidly OB individuals considering bariatric surgery (mean BMI = 48.8 kg/m2)	Total N=90 72.2% females	Morbidly OB individuals considering bariatric surgery N=90	NA	18- 66 (37.9± 11.2)	Childhood ADHD symptoms: retrospectively; WURS-k Adult ADHD symptoms: ADHD-SR	Binge eating: The EDE-Q; OBEs (eating an objectively large amount of food with a sense of loss of control)	The prevalence of childhood-onset ADHD was not different between individuals who screened positive or negative for BE (13.6% vs. 7.4%, p> .05)
Muller et al. (2014)	Cross-sectional	Bariatric surgery candidates and OB inpatients from a psychosomatic unit	Total N=156 72% females	Bariatric surgery candidates and OB inpatients Total N=156	NA	18–65 (39.91± 11.42)	Childhood ADHD symptoms: WURS-k Adult ADHD symptoms: ADHD-SR; CAARS; DSM-IV criteria	BED: Interview, BED module of the EDE; DSM-IV criteria	55.9% in Cluster 2 suffered from BED vs. 27.3% in Cluster 1 ($\chi^2(1) = 13.11$, p< .001); see Muller et al. (2014), Table 2, for Cluster 1 & Cluster 2 details Groups differed on the CAARS total score and on the inattention/memory, impulsivity and negative self-concept subscales (p< .05) but not on the hyperactivity subscale (p = .063)

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Table 4 (continued)

Reference (Year)/ Country	Type of Study/ Study Name	Participant Characteristics					ADHD Assessment	ED/Eating pathology assessment	Key Results	Confounder included in analysis (if any)
		Source Population	Sample size (% females)	Case Group (% females)	Control Group (% females)	Age Range (y)/ Mean Age (y) (mean \pm SD)				
Nazar et al. (2012) Brazil	Cross- sectional	Women seeking nonsurgical treatment of obesity	Total N=106 100% females	ADHD N=30 100% females	CG N=76 100% females		K-SADS module for ADHD, adapted for adults	BES	The ADHD group showed a significantly higher score when compared with the non-ADHD group in BES ($p < .01$)	
Nazar et al. (2014) Brazil	Cross- sectional	OB women seeking non-surgical treatment for obesity or eating disorders	Total N=132 100% females	OB ADHD N=40	OB only N=42 OB ED N=50	38.28 \pm 10.62	K-SADS module for ADHD, adapted for adults	BES	The OB ADHD group reported more BE symptoms than OB only and OB ED group ($p < .05$) Depressive symptoms , ADHD inattentive symptoms and trait impulsivity predicted BES scores ($p < .001$; $p < .001$; $p < .05$ respectively)	-
Nicolau et al. (2014) Spain	Case-control	Patients who underwent bariatric surgery	Total N=60 78.3% females	Patients who underwent bariatric surgery N=60	NA	46.3 \pm 9.8	Current adult ADHD symptoms: ASRS (18 items); DSM-IV criteria	The Revised Questionnaire of Eating and Weight patterns, self- report; Spanish version	The prevalence of BED was significantly higher in the ADHD group compared to CG (36.8% vs 14.6% , $p < .05$)	-

Pagoto et al. (2009) US	Cross-sectional CPES	Two nationally representative surveys of US adults	Total N=6,735 51.6% females	US residents N=6,735	NA	18-44 (31 ± 0.25)	Childhood ADHD: retrospectively; DIS for DSM-IV Adult ADHD symptoms: self-report; If a respondent met criteria for childhood ADHD, they were then asked whether problems persisted to the present; clinical reappraisal interviews were performed on a subsample of participants ADHD Symptoms: KSADS- PL; screen interview	BED; CIDI-the CIDI requires binges to persist only 3 months or more	Adult ADHD was significantly associated with past year BED (OR=5.1, 95% CI=2.83 - 10.72) Adults with ADHD had rates of past year BED (10.2%) that were six-fold that of adults with no history of ADHD (1.7%)	Stimulants/antidepressants medication, demographic and smoking status
Pauli-Pott et al. (2013) Germany	Cross-sectional	OW/OB children who had been referred for weight reduction	Total N=128 48% females	ADHD clinical symptoms N=17 ADHD Subclinical symptoms N=71	CG N=40	7.5-15 (11.0 ± 1.81)		BED; DSM-IV criteria; Structured interview-QEWP (parent, and adolescent version)	BED rates did not significantly differ between ADHD symptom groups (CG: 31.3%, Subclinical: 55.5%, Clinical: 13.3%; $\chi^2 = 1.69$, $p > .05$) No statistically significant interaction effects with sex on any of the eating problem scales	Sex, age, ODD/CD symptoms

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Table 4 (*continued*)

Reference (Year)/ Country	Type of Study/ Study Name	Participant Characteristics					ADHD Assessment	ED/Eating pathology assessment	Key Results	Confounder included in analysis (if any)
		Source Population	Sample size (% females)	Case Group (% females)	Control Group (% females)	Age Range (y)/ Mean Age (y) (mean \pm SD)				
Reinblatt et al. (2014) US	Retrospectiv e chart review	Two Pediatric Community Mental Health Clinics	Total N=252 47.2% females	ADHD N= 109 33.9% females	CG N=143 57.3% females	8-14 (10.8 \pm 3.7)	Clinical diagnostic evaluation; DSM-IV TR criteria The diagnostic procedure included interview with the child and parents/caregive rs and reviewing medical/psychia tric ratings, including ADHD scales when available	C-BEDS	The association between ADHD and BE symptoms was statistically significant (Crude Association OR = 16.13, p<. 001, 95% CI 4.75–54.73) and persisted after adjusting for relevant covariates	Comorbid diagnoses, medications, demographic variables, BN z-score, and clinic
Rosler et al. (2009) Germany	Cross- sectional	Female offenders	Total N=94 100% females	ADHD N=11	CG N=83	34 \pm 12	WRI; Retrospective assessment of childhood ADHD (WURS- k); diagnosis of ADHD in accordance to DSM-IV or ICD-10 research version (ADHD-DC)	SCID-I interview according to DSM- IV	Female offenders with ADHD were 6.2 times more likely to suffer from BED than non-ADHD controls (OR = 6.2, 95% CI: 1.5– 26.5)	-
Slane et al. (2010)	Cross- sectional	Twins recruited for the MSUTR	Total N=541 62%	Twins recruited for the MSUTR	NA	20.89 \pm 2.33	Only hyperactivity assessed; Hyperactivity	Minnesota Eating Behavior Survey- binge eating	Higher levels of hyperactivity were associated with greater levels	BMI

US			females	N=541				subscale of the Current Symptoms Scale-Self-Report Form	subscale	of BE (Model for BE, Pseudo R ² = 0.26, B= 0.13, p = .02). There was no effect of sex (p= .16)	
Sonneville et al. (2015)	Cohort study	Population based sample (women and their children)	Total N= 7,884	Population based sample N=7, 884	NA	Mid-adolescence: 14 and 16 yrs	Hyperactivity/inattention during mid- and late childhood: SDQ; teachers and parental reports	Participants asked to respond to 2 questions: how often during the past year they had eaten a very large amount of food, participants who reported positively (even occasionally) were asked a follow-up question about whether they felt out of control (yes/no) during these episodes	BES	Hyperactivity/inattention during late childhood indirectly predicted BE during mid-adolescence (standardized estimate 0.085, 95% CI: 0.007–0.128, p = .03) via late childhood overeating and early-adolescent strong desire for food	BMI during mid- and late childhood; se and maternal education
UK			7,120 analysed								
			48.8% females								
Steadman & Knouse (2014)	Cross-sectional	Undergraduate students from a private university in the Mid-Atlantic Region	Total N = 50	Students N = 50	NA	18-22	BAARS-IV			ADHD symptoms were significantly correlated with BE symptoms (r = 0.37, p < .01). None of the measures of impulsivity were found to be significant mediators between ADHD and BE symptoms (Bias-corrected CI for the Indirect effect included zero)	-
US			72% females								

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Table 4 (continued)

Reference (Year)/ Country	Type of Study/ Study Name	Participant Characteristics					ADHD Assessment	ED/Eating pathology assessment	Key Results	Confounder included in analysis (if any)
		Source Population	Sample size (% females)	Case Group (% females)	Control Group (% females)	Age Range (y)/ Mean Age (y) (mean \pm SD)				
Swanson et al. (2011) US	Cross- sectional	A representativ e sample of US adolescents	Total N = 10,123 48.7% females	A representativ e sample of US adolescents N = 10 123 48.7% females	NA	13-18	Parent self- report questionnaire (SAQ) based on DSM-IV criteria	Face to face interviews: Modified version of CIDI to assess AN, BN, BED based on DSM-IV criteria; sub- threshold EDs assessed using diagnostic algorithms	BED was not significantly associated with ADHD (OR = 2.1; 95% CI: 0.9- 4.6) SBED was significantly associated with ADHD (OR = 2.6; 95% CI: 1.4- 4.9; $p < .05$)	Age, sex, race/ethnicity

ADHD-DC = ADHD Diagnostic Checklist; ADHD-SR = ADHD Self-Rating scale; ALSPAC = Avon Longitudinal Study of Parents and Children; AN = Anorexia Nervosa; ASRS = The World Health Organization Adult AD/HD Self-Report Scale Adult Self Rating Scale; BAARS-IV = Barkley Adult ADHD Rating Scale-IV; BE = Binge Eating; BED = Binge Eating Disorder; BES = Binge Eating Scale; BN = Bulimia Nervosa; CAARS = Connors Adult ADHD Rating Scale; CAARS-S-SV = The Connors' Adult ADHD Rating Scale - report Screening Version; C-BEDS = Children's Binge Eating Disorder Scale; CD = conduct disorder; CG = Control Group; ChEAT = Children's Eating Attitudes Test; CIDI = Composite International Diagnostic Interview; CPES = The Collaborative Psych Epidemiology Surveys; CSI-4 = Child Symptom Inventory, 4th Edition; DIS = Diagnostic Interview Schedule; DSM-IV-TR = Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision; ED = Eating Disorder; EDE = Eating Disorder Examination; EDE-Q = The Eating Disorder Examination Questionnaire; EDO = Eating Disorders in Obesity Questionnaire; K-SADS = The Schedule for Affective Disorders and Schizophrenia; K-SADS-PL = Schedule for Affective Disorders and Schizophrenia for School-Age Children - Present and Lifetime Version; MSUTR = The Michigan State University Twin Registry; NFBC = Northern Finland Cohort; NW = Normal-weight; OB = Obese; OBEs = Objective Binge Eating Episodes; ODD = Oppositional Defiant Disorder; OW = Overweight; QE = Questionnaire on Eating and Weight Pattern; SAQ = Self-Administered Questionnaire; SBED = Sub-threshold Binge Eating Disorder; SCID = The Structured Clinical Interview for DSM Disorders; SCID-I = The Structured Clinical Interview for Diagnosis, according to DSM-IV Axis I Disorders; SDQ = Strengths and Difficulties Questionnaire; US = United States; WRI = Wender Reimherr Interview; WURS = Wender Utah Rating Scale; WURS-k = Wender Utah Rating Scale, German short version

Table 5 Studies presenting data relevant to an association between ADHD and LOC-eating

Reference (Year)/ Country	Type of Study/ Study Name	Participant Characteristics					ADHD Assessment	ED/Eating pathology assessment	Key Results	Confounds included in analysis (if any)
		Source Population	Sample size (% females)	Case Group (% females)	Control Group (% females)	Age Range (y)/ Mean Age (y) (mean \pm SD)				
Alfonsson et al. (2012) Sweden	Cross-sectional	OB patients presenting for bariatric surgery	Total N=187 73.3% females	Candidates for bariatric surgery N=187	NA	41.04 \pm 07	Adult ADHD: ASRS (18 items)	G-FCQ-T; LOC-eating subscale	Participants who screened positive for ADHD reported higher LOC scores vs. those who screened negative ($p < .001$). HADS anxiety and the ASRS impulsivity/hyperactivity subscale scores best explained scores on G-FCQ-T ($R^2 = 0.27$, $\beta = 0.38$, $p < .001$ and $\beta = 0.20$, $p = .012$, respectively)	-
Alfonsson et al. (2013) Sweden	Cross-sectional	OB patients presenting for bariatric surgery	Total N=245 72.7% females	Candidates for bariatric surgery N=245	NA	42.38 \pm 11.04	Adult ADHD: ASRS (18 items)	G-FCQ-T; LOC-eating subscale	Participants who screened positive for ADHD reported higher LOC scores vs. those who screened negative (women, $p = .013$; men, $p = .046$)	-
Erhart et al. (2012) Germany	Cross-sectional Part of BELLA study mental	A sub-sample of subjects from the KiGGS study's	Total N=2 414 49.3%	ADHD N=101 27.7% females	CG N= 2 313 50.2% females	11-17 ADHD: 11.98 \pm 3.19	German ADHD Rating scale (FBB-HKS/ADHS); DSM- IV criteria; parental	LOC-eating; SCOFF Questionnaire (1 item): 'Do you worry you have lost control over	For each weight group a higher proportion of responders with ADHD reported LOC ($\chi^2 =$	Weight status: age, sex, and SES

	health module of the KiGGS	national representative sample	females			CG:	report	how much you eat?	116.693; p< .001)	
Pagoto et al. (2010)	Cross-sectional	OB patients who completed a 16-week behavioral weight loss program	Total N=63 75% females	ADHD N= 19 (30% of total sample)	CG N=44	12.68±3.21 49 ± 10.3	Adult ADHD; ASRS (18-item)	Self-efficacy to control eating; WEL	Individuals who screened positive for ADHD reported lower self-efficacy (F (1, 62) = 12.46, p=.001) vs. those who screened negative	Sex, age
US										
Reinblatt et al. (2015)	Cross-sectional	Participants were referred from the community, through pediatric mental health clinics, medical clinics, and flyers on public bulletin boards	Total N=79 48% females	Children with ADHD N= 44 (8 Non-OW/OB; 36 OW/OB)	CG N= 35 (17 Non-OW/OB; 18 OW/OB)	8-14	K-SADS-PL ADHD symptoms; CPRS-R ADHD diagnosis: K-SADS-PL diagnosis of current ADHD and a T-score >65 on either or both of the CPRS-R DSM-IV-TR Scales (Inattentive and/or Hyperactive-Impulsive)	Loss of Control-Eating Syndrome (LOC-ES): CheDE & SPEEI	The odds of LOC-ES were 12.68 times higher in children with ADHD vs. controls (OR = 12.68, 95% CI: 3.11-51.64, p < .001). A 5-point higher T-score on the Inattentive subscale was associated with 1.39 times higher odds of LOC-ES (OR = 1.39, 95% CI: 1.14-1.70, p = .001). A 5-point higher Hyperactivity/Impulsivity T- score (OR =1.41, 95% CI: 1.15-1.73, p = .001) was associated with a significantly greater likelihood.	Age, sex, race and BMI z-score
US										

ASRS = The World Health Organization Adult AD/HD Self-Report Scale Adult Self Rating Scale; CG = Control Group; ChEDE =Eating Disorder Examination adapted for Children; CPRS-R = The Revised Conners' Parent Rating Scale; FBB-HKS/ADHS = The German ADHD Rating scale - The FBB-HKS/ADHS is part of the comprehensive Diagnostic System for Mental Disorders in Childhood and Adolescence; G-FCQ-T = General-Food Cravings Questionnaire-Trait; KiGGS = the German Health Interview and Examination Survey for Children and Adolescents; K-SADS-PL = Schedule for Affective Disorders and Schizophrenia for School-Age Children–Present and Lifetime Version; LOC = Loss of control over Eating; LOC-ES = Loss of control Eating syndrome; OB = Obese; OW = Overweight; SPEEI = Standard Pediatric Eating Episode Interview

Table 6 Studies presenting data relevant to the association between ADHD and overeating behaviour

Reference (Year)/ Country	Type of Study/ Study Name	Participant Characteristics					ADHD Assessment	ED/Eating pathology assessment	Key Results	Confounders included in analysis (if any)
		Source Population	Sample size (% females)	Case Group (% females)	Control Group (% females)	Age Range (y)/ Mean Age (y) (mean \pm SD)				
Alfonsson et al. (2012) Sweden	Cross-sectional	OB patients presenting for bariatric surgery	Total N=187 73.3% females	Candidates for bariatric surgery N=187	NA	41.04 \pm 07	Adult ADHD: ASRS (18 items)	G-FCQ-T; Emotional craving subscale	Participants who screened positive for adult ADHD reported higher scores of emotional craving vs. those who screened negative (p = .002)	-
Alfonsson et al. (2013) Sweden	Cross-sectional	OB patients presenting for bariatric surgery	Total N=245 72.7% females	Candidates for bariatric surgery; N=245	NA	42.38 \pm 11.04	Adult ADHD: ASRS (18 items)	G-FCQ-T; Emotional craving subscale	Participants who screened positive for adult ADHD reported higher scores of emotional craving vs. those who screened negative (p = .001)	-
Davis et al. (2006) Canada	Cross-sectional	Adult women recruited from community settings	Total N=110 100% females (47% Normal Weight, 21% OW,	Adult women N=110	NA	25-46 (33.3 \pm 6.7)	Childhood ADHD: Retrospectively; WURS	Emotional & external eating: The DEBQ Binge eating: BEQ Emotional Overeating: The EES –Depression subscale	ADHD symptoms positively related to forms of overeating and overeating, positively correlated with BMI; model was a good fit to the data (χ^2 = 15.22, df = 13, p	

Dempsey et al. (2011)	Cross-sectional	OB individuals recruited from community settings	30% OB) Total N= 125 65.6% females	≥ 4 symptoms of ADHD N=27	<4 symptoms of ADHD N=98	43.7 ± 13.47	Adult ADHD: ASRS–VI.1 (6 items)	Disinhibition and hunger: EI Emotional eating: EES	= .294) Adults who had ≥4 current ADHD symptoms were more disinhibited around food (p = .03), more susceptible to their own perceptions of hunger (p = .03), and reported higher scores in emotional eating (p = .00) vs. adults who had <4 ADHD symptoms	-
Hartman et al. (2013)	Experimental	Participants recruited from community settings	Total N=90 48.8% female	ADHD Group N=33 33.33% females	CG: N=33 54.5% females LOC Group: N= 24 62.5% females	10-14	SUB ADHD; DSM-IV-TR, minimum of 4 diagnostic criteria in at least one of the diagnostic sections. Parental reports	Ad libitum snack food intake was assessed in a 10-min trial during which the participants was asked to watch an age-appropriate movie Parental report:	The ADHD group consumed more snack food than the other groups (ADHD vs. control; p< .01; ADHD vs. LOC; p< .05) ADHD had a direct positive effect on consumption of unhealthy food (β = 0.202, p < .001) and on bulimic dietary behaviours (β = 0.31, p< .001)	Hunger, liking of foods, representativ eness of eating behaviour
Kim et al. (2014)	Cross-sectional	Population-based; children from 27 elementary schools in Cheonan, the Republic of Korea	Total N= 12,350 51.3% females	Children from Cheonan N= 12,350	NA	5-13 9.4 ± 1.7	DuPaul ADHD Rating Scale; DSM-IV diagnostic criteria	Dietary behavior consisted of overeating and diet speed compared with other families (slower, similar, faster)		-

(continued on next page)

Table 6 (continued)

Participant Characteristics										
Reference (Year)/ Country	Type of Study/ Study Name	Source Population	Sample size (% females)	Case Group (% females)	Control Group (% females)	Age Range (y)/ Mean Age (y) (mean \pm SD)	ADHD Assessment	ED/Eating pathology assessment	Key Results	Confounders included in analysis (if any)
Munsch et al. (2011) Switzerland	Experimenta l	OW children and their mothers who were recruited to participate in a treatment trial for OW and OB	Total N=41 56.1% females	Total sample N=41	NA	8-12	Attention problems: parental assessment of the CBCL	Taste test	Children with higher scores for attention problems consumed larger amounts of food (standardized path coefficient =.45, $p < .05$)	
Pagoto et al. (2010) US	Cross- sectional	OB patients who completed a 16-week behavioral weight loss program	Total N=63 75% females	ADHD Group N= 19 (30% of total sample)	CG N=44	49 \pm 10.3	Adult ADHD; ASRS (18-item)	Emotional eating: WALI-emotional eating scale	Individuals who screened positive for ADHD reported higher levels of emotional eating vs. those who screened negative ($F(1, 62) = 10.07$, $p = .002$)	Sex, age
Patte et al. (2016) Canada	Cross- sectional	Community- based sample	Total N = 421 74.8% females	Community- based sample N= 421 74.8% females	NA	24-50 (33.56 \pm 6.66)	Childhood ADHD: Retrospectively; WURS Adult ADHD: CAARS-S-SV	Binge eating: BEQ Emotional eating: DEBQ Hedonic eating: PFS	Overeating mediated the ADHD symptoms-BMI association ($p < .0001$). There were no sex differences on any of the parameter categories	Age, ethnicity, education

Pauli-Pott et al. (2013) Germany	Cross-sectional	OW/OB children who had been referred for weight reduction	Total N=128 48% females	ADHD clinical symptoms N=17 Subclinical symptoms N=71	CG N=40	7.5-15 (11.0 ± 1.81)	ADHD Symptoms: KSADS- PL; screen interview	External and emotional eating: EPI; self-report	ADHD symptom groups did not differ with respect to external and emotional eating (p> .05)	Sex, age, ODD/CD symptoms
Strimas et al. (2008) Canada	Cross-sectional	Adult men recruited from community settings	Total N=145 0% females (34.5% NW, 33.1% OW, 32.4% OB)	Adult men N=145	NA	25-50 (34.42 ± 7.7)	Childhood ADHD: Retrospectively; WURS Adult ADHD: CAARS-S-SV	Emotional and external eating: DEBQ Binge eating: BEQ Emotional Overeating: EES – Depression subscale	ADHD symptoms positively predicted overeating in males, which in turn positively predicted BMI; model was a good absolute fit to the data ($\chi^2 = 26.30$, $df = 19$, $p = .123$)	-
Wilhelm et al. (2011) Germany	Experimental 1 2 x 2 factorial design	Children with ADHD: clinical sample; admitted to inpatient or outpatient treatment Controls: non-ADHD recruited from the same site (Hospital via advertisements)	Total N=94 0% females	Lean ADHD (ADHD+O W-): N=24 OW ADHD (ADHD+O W+): N=22	Lean without ADHD (ADHD-OW-): N=27 OW without ADHD (ADHD+OW +): N=21	7-15	K-SADS-PL ADHD symptom severity: German Parental and Teacher Report on ADHD symptoms (FBB-HKS)	Food intake during a standardised ‘‘test’’ buffet style breakfast meal	Food intake (kcal) did not differ among the 4 groups ($p > .05$) The interaction effect between diagnostic group and weight status was not significant, ($F(7,83) = 0.44$, $p = .87$)	Medication (mg per kg body weight)

ASRS = The World Health Organization Adult AD/HD Self-Report Scale Adult Self Rating Scale; BEQ = The Binge Eating Questionnaire; CAARS-S-SV = The Conners' Adult ADHD Rating Scale –Self-report Screening Version; CBCL = Child Behavior Checklist; CD = conduct disorder; CG = Control Group; DEBQ = The Dutch Eating Behaviour Questionnaire; DSM-IV-TR = Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision; EES = The Emotional Eating Scale; EI = Eating Inventory; EPI = Eating Pattern Inventory; G-FCQ-T = General-Food Cravings Questionnaire-Trait; KSADS- PL = Schedule for Affective Disorders and Schizophrenia for School-Age Children–Present and Lifetime Version; LOC = Loss of control over Eating; NW = Normal-Weight; OB= Obese; ODD = Oppositional Defiant Disorder; OW = Overweight; PFS = The Power of Food Scale; SUB = Subthreshold; WALI = Weight and Lifestyle Inventory; WURS = Wender Utah Rating Scale

Table 7 Associations between specific symptoms of ADHD and ED subtypes and/or disordered eating behaviour

ED/ Disordered eating behaviour	Symptoms of ADHD									
	Hyperactivity		Impulsivity		Inattention		Hyperactivity/ inattention		Hyperactivity/ impulsivity	
	YES	NO	YES	NO	YES	NO	YES	NO	YES	NO
BN/ BN symptoms		Mikami et al. 2008 Mikami et al. 2010 Cortese et al. 2007 Seitz et al. 2013	Mikami et al. 2008 Mikami et al. 2010 Cortese et al. 2007	Seitz 2013	Seitz et al. 2013 Cortese et al. 2007 Yates et al. 2009	Mikami et al. 2008 Mikami et al. 2010		Grabarek & Cooper 2008		
BED/ BED symptoms	Slane et al. 2010	Muller et al. 2014	Muller et al. 2014		Nazar et al. 2014 Muller et al. 2014	Davis et al. 2009	Sonneville et al. 2015	Khalife et al. 2014	Goldschmidt et al. 2015	Nazar et al. 2014 Davis et al. 2005
Binging & purging LOC, emotional craving					Yates et al. 2009 Bleck et al. 2015 Reinblatt et al. 2015	Alfonsson et al. 2012			Bleck et al. 2015 Reinblatt et al. 2015 Alfonsson et al. 2012	
AN Restrictive Subtype Restrictive eating						Yates et al. 2009 Rastam et al. 2013 Bleck et al. 2015			Rastam et al. 2013, in boys	Bleck et al. 2015
Dietary restraint	Slane et al. 2010 (stronger in men)									
Drive for thinness	Grabarek & Cooper 2008, in men					Grabarek & Cooper 2008, in men				
Snack frequency							Ebenneger et al. 2012			
Snack Intake			Wilhelm et al. 2011		Munsch et al. 2011	Wilhelm et al. 2011				
Weight preoccupation		Slane et al. 2010								
Eating, shape, weight concern		Muller et al. 2014	Muller et al. 2014		Muller et al. 2014					